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THE DIAGNOSIS
OF
NERVOUS DISEASES

BY

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PREFACE

It is seldom in clinical medicine that we meet with diseases in their typical forms, at least in the forms described in text-books; more often we have to deal with patients who exhibit signs and symptoms common to several diseases. The present volume approaches the subject of diagnosis of nervous diseases from the clinical standpoint, avoiding abstruse details of purely theoretical interest.

The subject of the work is mainly that of diagnosis; treatment is not discussed, save incidentally here and there. After a short introductory account of the more important facts in the anatomy and physiology of the nervous system, the various signs and symptoms of nervous diseases are discussed and illustrated by clinical cases. The order pursued is that in which it is convenient to study the phenomena during the process of diagnosis.

All the clinical illustrations are taken from cases which I have personally observed, most of them at the Westminster Hospital.

I hope that the book, despite imperfections of which I am conscious, and of which many are due to the original lecture form, will prove useful to the advanced student and to the practitioner, not to replace but to supplement the text-books already in use.

The lectures of which the work consists were delivered, some to undergraduate audiences at the Westminster Hospital, others to postgraduates in London and elsewhere. A few have already appeared in various medical journals—the *Practitioner*, the *International Clinics*, and the *Edinburgh Medical Journal*, to the publishers of which I gladly express my acknowledgments.

To many of my colleagues on the staff of the Westminster Hospital I am indebted for their courtesy in permitting me to study their cases. My thanks are due to various friends for photographs of cases, many of which were taken by themselves. Amongst these I would particularly mention Dr. James Collier (Figs. 29 and 116), Mr. P. Lockhart Mummery (Fig. 121), Dr. A. Ross Sinclair (Fig. 66), Dr. H. R. Sedgwick (Fig. 55), Dr. A. H. Watt (Fig. 140), and Dr. S. J. Aarons (Fig. 22). Dr. F. X. Dercum, Dr. M. Allen Starr, and Mr. Harold Barwell have kindly allowed me to use certain of their diagrams, to which their names are attached in the text. I am also indebted to the publishers of the *Practitioner* for the use of a number of photographic illustrations in the lectures on hysteria and on affections of the cervical sympathetic, originally published in that journal.

Lastly, I desire to express my grateful thanks to my friend Dr. A. F. Street for his invaluable help in the correction of the proof-sheets as they passed through the press.

PURVES STEWART.

HARLEY STREET, LONDON, W.
September 1906.

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THE DIAGNOSIS OF NERVOUS DISEASES

LECTURE I

ANATOMY AND PHYSIOLOGY

There is no department of medicine where an accurate knowledge of anatomy is of greater importance than in the diagnosis of nervous diseases. Let us therefore, at the outset of this series of lectures, recall some of the main points in the anatomy and physiology of the nervous system.

The nervous system consists of two main divisions:—(1) the cerebro-spinal, comprising the brain and spinal cord, together with the cranial and spinal nerves, and (2) the sympathetic, constituted by two chains of pre-vertebral ganglia, one on each side of the spine. These two, the cerebro-spinal and sympathetic, intercommunicate.

For teaching purposes it is convenient to regard the nervous system as built up of nerve-cells, and their processes the nerve-fibres. Both are excitable. But whereas the nerve-cell has been commonly assumed to originate impulses as does the cell of an electric battery, the nerve-fibres serving merely as conductors, it is more than doubtful whether any impulse ever arises within a nerve-cell, except as the result of a transmitted impulse. Each nerve-fibre is made up of a bundle of extremely fine neuro-fibrillæ which traverse the nerve-cell, entering it through one process and leaving it through another. In this way the nerve-cell acts as a convenient shunt for impulses, receiving them from one quarter and transmitting them to another. The nerve-cell also exerts a trophic influence over the nerve-fibre and is intimately concerned with its nutrition, so that if separated from its trophic nerve-cell the nerve-fibre degenerates.

In a **reflex motor act**, which is the simplest manifestation of nervous energy, as for example in the plantar reflex, the impulse or stimulus starts from a sensory end-organ, in this instance the skin of the sole. The impulse travels up a sensory nerve-fibre, through the corresponding posterior nerve-root into the spinal cord, and there, through the intermediation of another inter-communicating nerve-fibre and cell in the grey matter of the cord, it reaches an anterior cornual cell. From this motor-cell an efferent impulse starts, travelling outwards along an anterior nerve-root into a peripheral nerve and thus reaches a muscle-fibre in the flexor muscle of the toes. The muscle contracts and withdraws the sole of the foot from the original irritant.

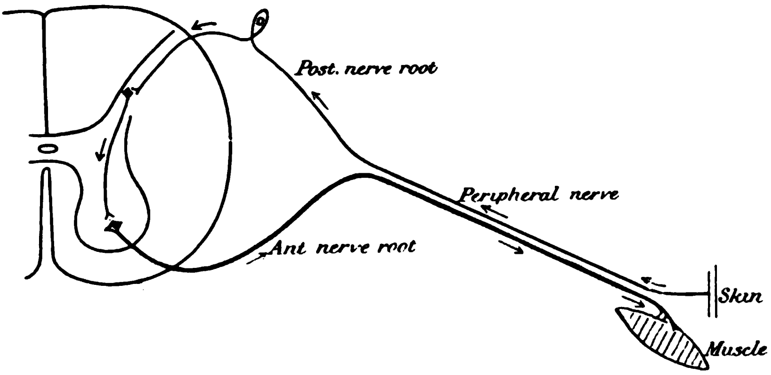

FIG. 1.—Diagram of a Simple Spinal Reflex.

The accompanying diagram (Fig. 1) will serve to recall the chief components of a simple spinal reflex, such as we have just described.

Some reflexes occur unconsciously, as for example the reflex contraction of the pupil when the retina is stimulated by light, or again, the normal movements of the stomach and intestines. But in other cases the afferent impulse, besides exciting a reflex motor action, sends part of its impulse upwards to the higher centres of the opposite cerebral cortex, where it produces a conscious sensation. This is accomplished by means of a sensory fibre passing upwards in the substance of the spinal cord, as indicated in the other diagram (Fig. 2), through relays of nerve-cells and fibres in the medulla, pons, and so on, to the perceiving centre in the cortex.

Moreover, a discharge of motor energy from the motor cell in the anterior cornu can be produced not only reflexly, from below, but also voluntarily, from above. This is achieved by an impulse descending from the cortical motor centre of the opposite side, through the crossed pyramidal tract down the cord to the anterior cornual cell (see Fig. 2). A discharge can also be voluntarily inhibited from above.

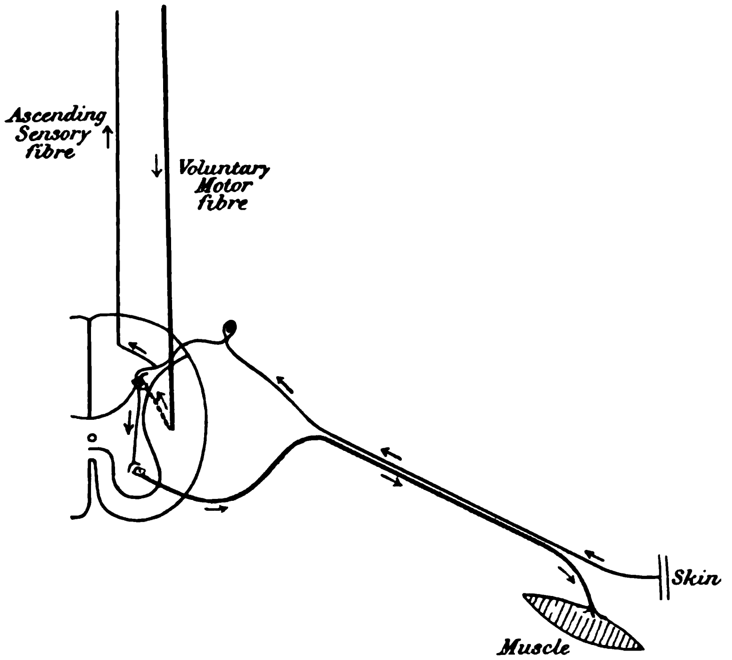

FIG. 2.—Diagram of a Voluntary Motor Act.

Confining ourselves now to the consideration of a cerebro-spinal reflex motor action, we must bear in mind that afferent impulses, on reaching the cortical sensory centres, do not necessarily produce a descending impulse along the pyramidal tract. If they do, this is simply an automatic action, or a longer variety of reflex. There exist in the cortex perceiving-centres which take cognisance of the source and nature of the stimulus, and determine whether or not any active notice shall be taken of it, that is to say, whether a voluntary (and not merely an automatic) movement shall or shall not take place.

Some reflexes, even though associated with conscious sensory impressions (for example vomiting, or the sexual reflex), cannot be inhibited. This is possibly owing to the absence of antagonistic muscles which could prevent the reflex. But other reflexes can be inhibited by contracting the opposing muscles and thereby fixing the part which would otherwise make a reflex movement.

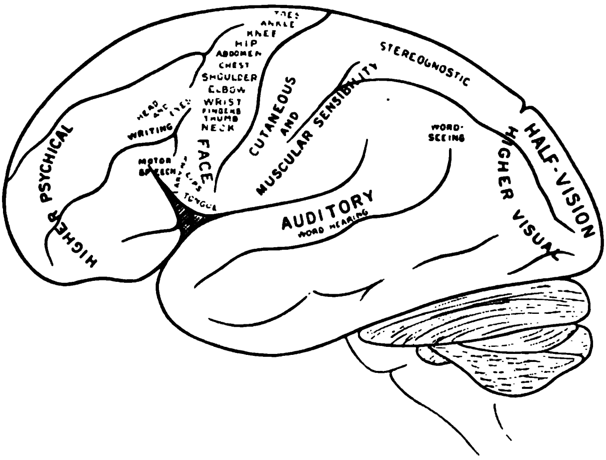

FIG. 3.—Scheme of Cerebral Localisation (outer surface).

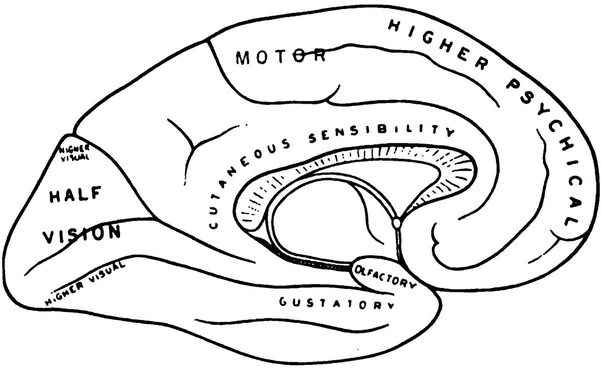

FIG. 4.—Scheme of Cerebral Localisation (mesial surface).

Finally, by education, a motor impulse can be initiated at the cortical motor centre, without any preceding afferent impulse from the part to be moved. All movements in a new-born infant are either reflex or automatic, and only gradually does the child learn to call in antagonistic muscles, and, by an effort of will, to inhibit reflex acts and to initiate voluntary ones.

Certain more complicated reflexes, such as the reflex movements of respiration, have their centres in the medulla; others, such as the movements of the heart and blood-vessels, have their lower reflex centres in the sympathetic ganglia, but can also be influenced by the cerebro-spinal nervous system. Others again, such as the movements of the stomach and intestine, can be performed independently of the central nervous system.

Figures 3 and 4 are diagrams of the cerebral cortex, both on its convex and its mesial aspect. It is unnecessary here to enumerate in detail the various fissures and sulci, or the different lobes and convolutions.

When we look at a brain, the first landmarks to be identified are the Sylvian and Rolandic fissures. The fissure of Rolando starts at the middle line above, from a point half an inch behind the mid-point between the glabellum and the external occipital protuberance. It runs downwards and forwards, along the convex surface of the brain, in the direction of the anterior part of the Sylvian fissure, making an angle of about 67½ degrees, *i.e.* three-quarters of a right angle, with the middle line.

These two figures also show diagrammatically our present views on cerebral localisation. It should be particularly observed that the motor areas on the convex surface extend back as far as the Rolandic fissure but not behind it, as was formerly taught. Not only by experimental stimulation in anthropoid apes,[1] and in certain cases in man, but also by histological research,[2] it has been shown that the posterior wall of the Rolandic fissure differs in structure and function from the anterior or motor wall. The anterior wall possesses giant pyramidal cells with no granular layer, whilst the posterior wall has a distinct granular layer with no giant cells.

---

[1] Sherrington and Grünbaum, *Trans. Path. Soc. Lond.*, 1902, vol. liii. p. 127.

[2] Campbell, A. W., *Histological Studies on the Localisation of Cerebral Function*, 1905.

Brodmann, K., *Journ. f. Psychologie und Neurologie*, Bd. ii. p. 80

Another point to remember is that the different so-called motor areas are not sharply marked off from one another, as the diagram might indicate, but they overlap, each area signifying that stimulation of that point produces the maximum movement of the particular part mentioned.

The chief path by which motor impulses are conducted from the cortical motor areas to the muscles, is the pyramidal tract, whose course is diagrammatically indicated in Fig. 5. From the motor cells in the cortex the fibres converge through the *corona radiata* into the great strand of nerve-fibres between the lenticular nucleus externally and the optic thalamus and caudate nucleus internally, namely, the internal capsule. Fig. 6 shows a horizontal section through the internal capsule, in which we notice that it has an anterior and a posterior limb, joining each other at an obtuse angle, the *genu* or knee. The motor fibres for the leg and arm occupy the anterior two-thirds of the posterior limb, the fibres for the tongue and mouth are at the genu, those for the face just in front. But the order in which these different strands pass through the internal capsule is not quite the same as that in which they started from the cortex. Thus we notice that immediately behind the fibres for the lips we have, from before backwards, those for the shoulder, elbow, and fingers, then for the trunk, and lastly for the hip, knee, and toes. We also notice, in passing, that the pathway of the sensory fibres traverses the posterior part of the capsule, and that behind them again are the visual fibres. The sensory fibres probably do not form a separate, compact bundle, but are partially mingled with some of the motor fibres for the leg.

Before leaving Figs. 5 and 6, it is of interest to study briefly, with their help, the different effects produced by lesions of the pyramidal motor tract at various levels.

A lesion in or near the motor cortex, if moderate in size, will produce, according to its situation, a monoplegia of the face, arm, or leg, on the opposite side of the body. A somewhat larger lesion will produce a brachio-facial or a brachio-crural monoplegia. (We

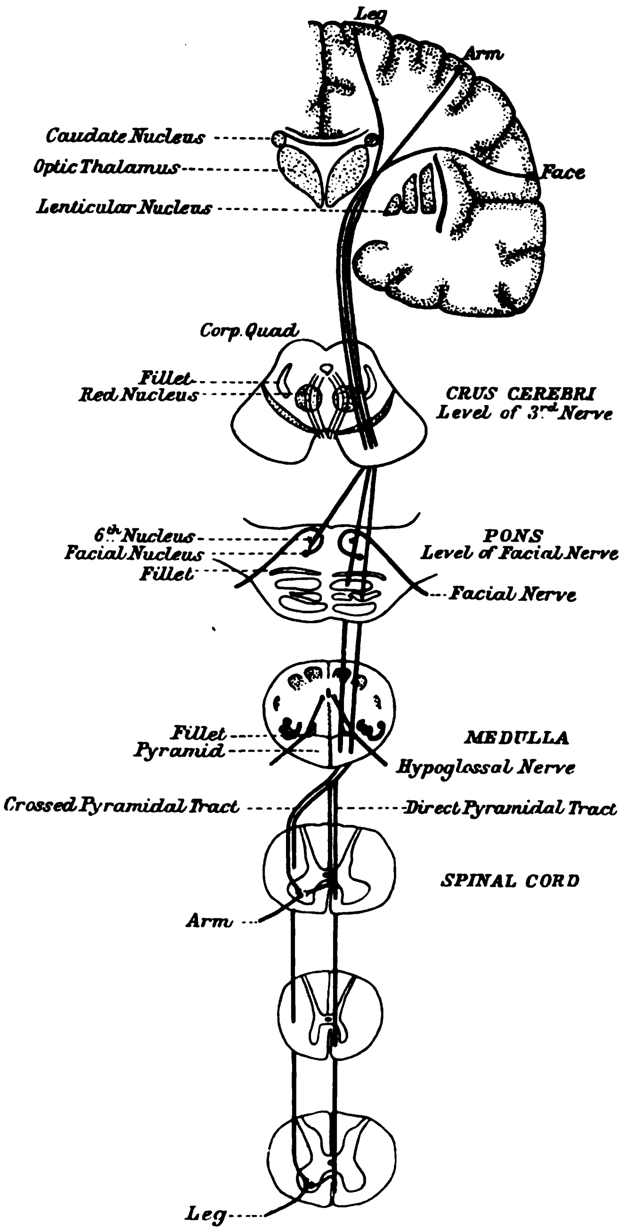

FIG. 5.—Diagram of Pyramidal Tract and its course through the brain and cord.

observe that facio-crural monoplegia without implication of the arm is impossible from a single lesion.) For the production of a complete hemiplegia of face, arm, and leg there must be a very extensive cortical lesion. But in the *internal capsule* all these strands are closely crowded together, so that a moderate-sized capsular lesion can produce a complete hemiplegia, whilst a capsular lesion small enough to cause a mere monoplegia is well-nigh impossible.

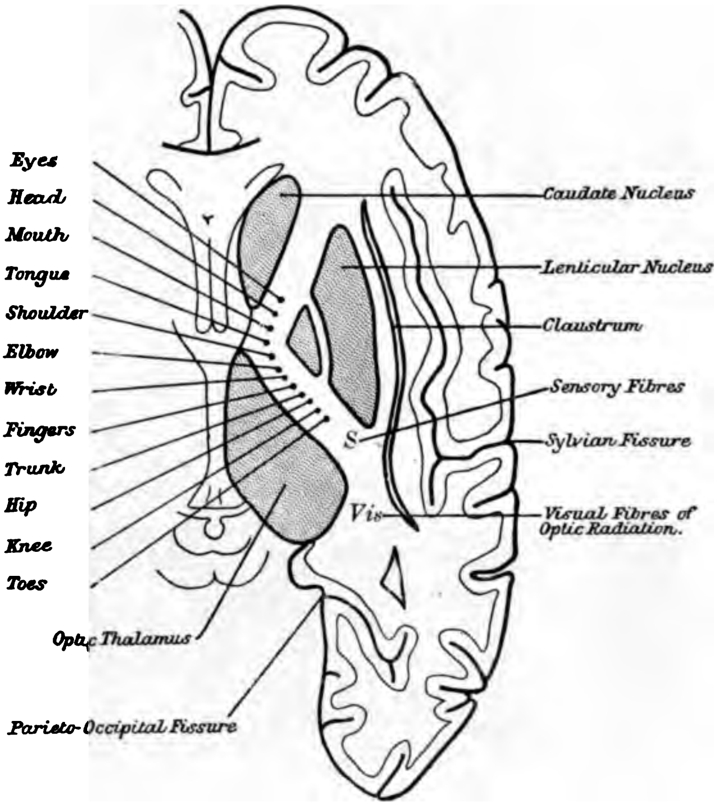

FIG. 6.—Horizontal section through right cerebral hemisphere, showing position of the various strands in the internal capsule. (After Beevor and Horsley.)

If the capsular lesion be in the region of the *genu* we have hemiplegia of face, arm, and leg. And moreover, from paralysis of the muscles which rotate the head and eyes to the opposite side, the patient has "conjugate deviation" of the head and eyes towards the side of the lesion, owing to unopposed action of the muscles supplied by the intact hemisphere.

If, on the other hand, the capsular lesion be farther back along the posterior limb of the capsule, the hemiplegia will affect the leg much more than the arm, and the face only slightly; whilst, owing to interference with the sensory tract, there will now be hemianæsthesia also.

Lastly, if the lesion be at the extreme posterior end of the capsule, there will be not only hemianæsthesia but also hemianopia from interruption of the visual fibres. Here again we note that it is impossible for a single capsular lesion to produce at the same time hemiplegia and hemianopia without also producing hemianæsthesia.

A lesion of the crus cerebri will tend to implicate the third cranial nerve on the side of the lesion, producing at the same time a hemiplegia of face, arm, and leg on the opposite side. This so-called "Weber's syndrome" is one variety of "crossed paralysis."

A unilateral lesion of the pons at the level of exit of the facial nerve will produce another "crossed paralysis," viz.:—facial palsy on the side of the lesion with hemiplegia of the arm and leg of the opposite side. And if at the same time the nucleus of the sixth cranial nerve be implicated, (which is not unusual, since the facial nerve hooks around the sixth nucleus within the pons), we have nuclear palsy of the sixth nerve, facial palsy on the same side, and hemiplegia of the arm and leg on the opposite side:—the "Millard-Gubler syndrome."

Unilateral lesions of the pons or medulla below the level of the facial nerve leave the face unaffected and produce only a hemiplegia of arm and leg. And a *unilateral lesion of the spinal cord* below the cervical enlargement will produce a monoplegia of the leg on the side of the lesion without affection of the arm. It will also produce some anæsthesia of the opposite leg. Such motor paralysis of one leg and sensory paralysis of the other is called "Brown-Séquard paralysis," to which we shall return later.

Fig. 7 is a diagram representing the tracts in the spinal cord of chief clinical importance. There are also other ascending and descending tracts of minor importance, which we have omitted from the diagram for the sake of simplicity.

The pyramidal tracts are by far the most important descending tracts in the cord, for they convey voluntary motor impulses downwards from the motor cortex towards the anterior cornua. The pyramidal fibre does not actually join the anterior cornu, but ends in the region of the posterior cornu, whence a short intermediate connecting-cell and fibre run forward, linking it to the anterior cornual cell (see Fig. 2). Most of the voluntary motor impulses decussate at the lower end of the medulla and traverse the crossed pyramidal tract in the lateral column, a few run in the direct pyramidal tract and cross over later within the cord itself. A few pyramidal fibres also run down in the homolateral pyramidal tract (which we might, somewhat paradoxically, call the uncrossed crossed-pyramidal tract).

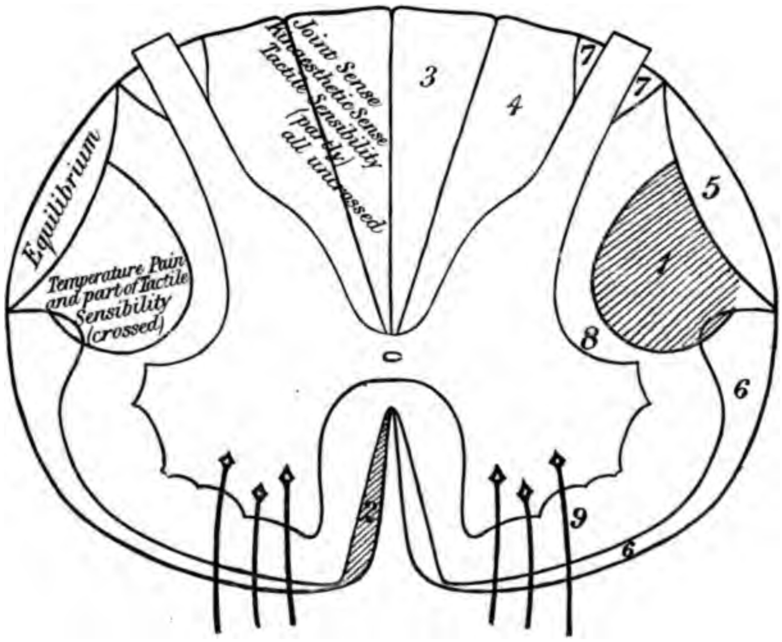

FIG. 7.—Diagram of Tracts in the Spinal Cord.

1. Crossed pyramidal tract.
2. Direct pyramidal tract.
3. Postero-internal tract (column of Goll).
4. Postero-external tract (column of Burdach).
5. Direct or dorsal cerebellar tract.
6. Ventral cerebellar tract (Gowers).
7. Lissauer's marginal zone.
8. Lateral ground-bundle
9. Anterior ground-bundle.

These fibres probably do not cross over, but supply motor impulses to the homolateral leg.

Sensory Paths.—All the sensory impulses, whether conveying sensations of touch, temperature, or pain from the skin, sensation of active muscular contraction from the muscles (kinæsthetic sense), or sensations from the joints or bones, enter the spinal cord through the posterior roots, as indicated in Fig. 8. Of these various impulses, the fibres conveying sensations from the muscles and joints, together with the smaller part of the fibres for tactile sensation, ascend, uncrossed as yet, in the posterior column of the same side, to the gracile and cuneate nuclei of the medulla.

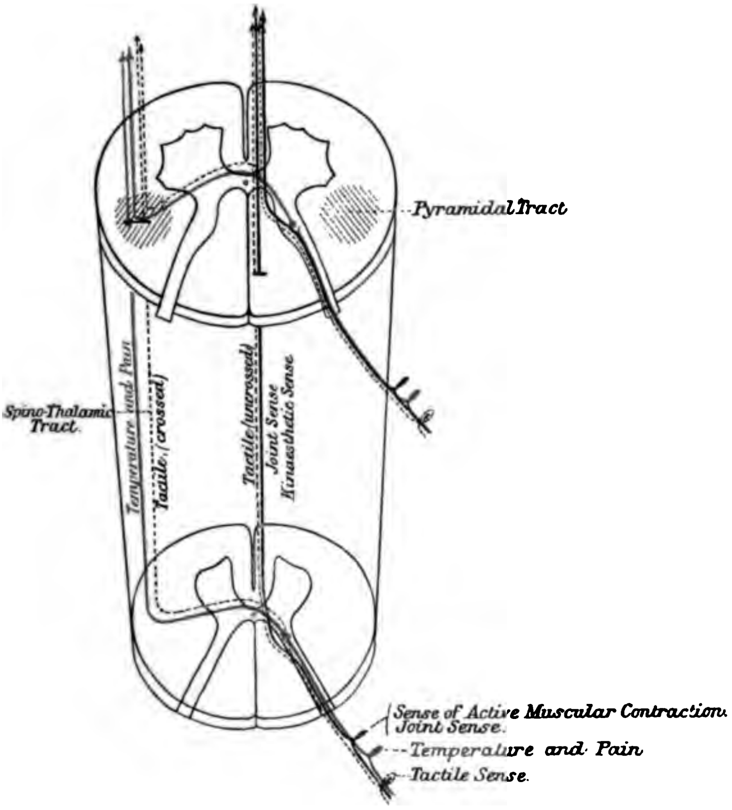

FIG. 8.—Diagram illustrating the course of the various sensory paths in the spinal cord.

Most of the fibres for tactile sense, together with those for temperature and pain, cross in the anterior commissure to the opposite side (these crossed fibres not coming directly from the posterior root, but through the relay of another cell in the posterior horn) and ascend the cord in the opposite lateral column, in the spino-thalamic tract. Thus the lateral column conducts not only pyramidal motor impulses coming down, but also spino-thalamic ascending impulses of touch, temperature and pain.

The upward course of the various sensory fibres through the medulla and pons is somewhat complicated, and not yet entirely settled, but the most probable arrangement is shown in Fig. 9.

Most of the fibres for *touch* cross the middle line in the spinal cord, as already explained, and then pass directly upwards in the spino-thalamic tract of the lateral column and into that part of the medulla called the *formatio reticularis.* The formatio reticularis leads the fibres up through the pons and crus to the optic thalamus, and thence they pass through the posterior limb of the internal capsule to the sensory cortex behind the fissure of Rolando.

As the sensory tract traverses the pons it passes along the inner side of the sensory spinal root of the trigeminal nerve of the same side. Thus a *unilateral lesion of the formatio reticularis* below the exit of the fifth or trigeminal nerve will produce a "crossed anæsthesia," *i.e.* anæsthesia of the face on the side of the lesion, and of the arm, leg, and trunk of the opposite side. But higher up the pons the sensory fibres from the face also cross the middle line, so that a lesion of the formatio reticularis in the crus cerebri will now cause complete hemianæsthesia of face, arm, and leg (see Fig. 10).

The fibres for *temperature and pain*, entering like the others by the posterior root, pass into the grey matter of the posterior cornu. There they start afresh and cross to the opposite side of the cord, ascending in the opposite lateral column near the crossed tactile fibres. On reaching the medulla, they diverge from the tactile fibres and pass to the outer side of the olivary body, close to the lateral margin of the medulla and intermingled with the fibres of Gowers' tract. They then leave the region of Gowers'

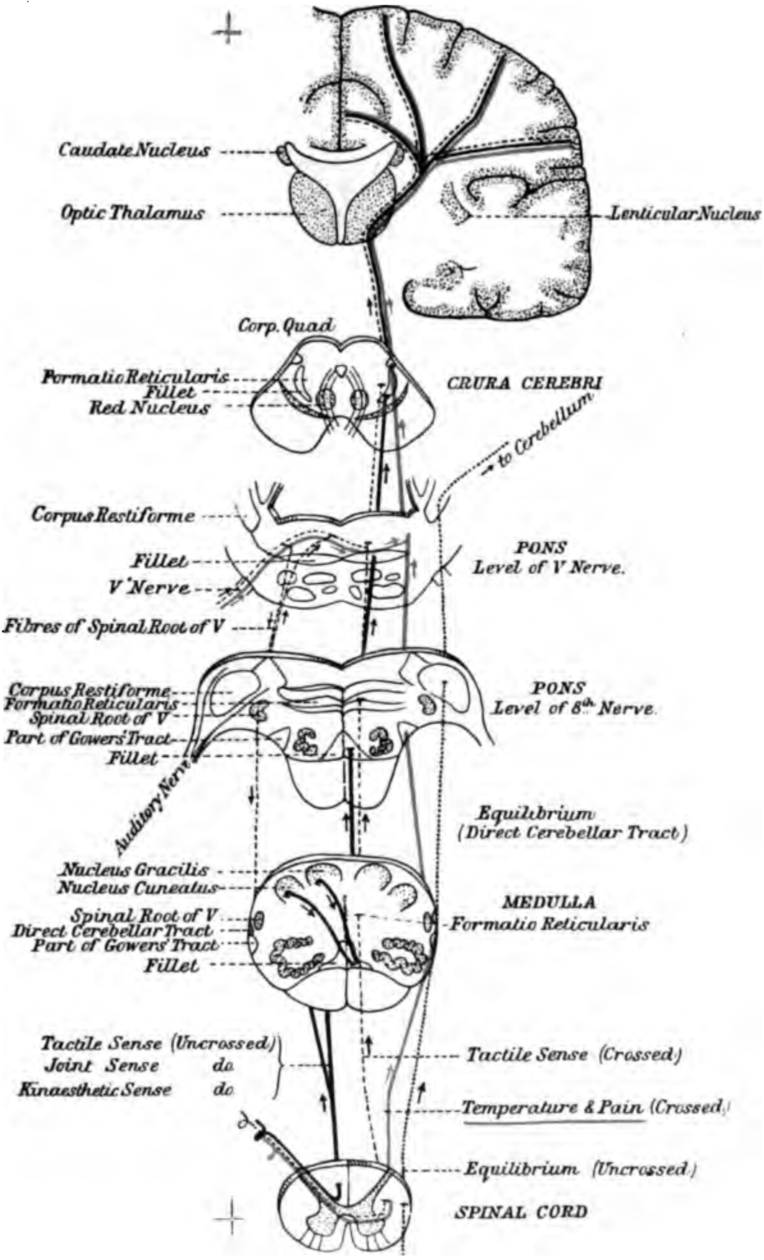

FIG. 9.—Diagram of Chief Sensory Tracts in Spinal Cord, Medulla, Pons, and Cerebrum.

tract and pass upwards through the pons, gradually inclining towards the other sensory tracts and ultimately ascend with them to the optic thalamus and thence through the internal capsule to the cortex. Gowers' tract, now separate from the temperature and pain fibres in the upper part of the pons, suddenly hooks backwards and enters the cerebellum from above, through the superior peduncle.

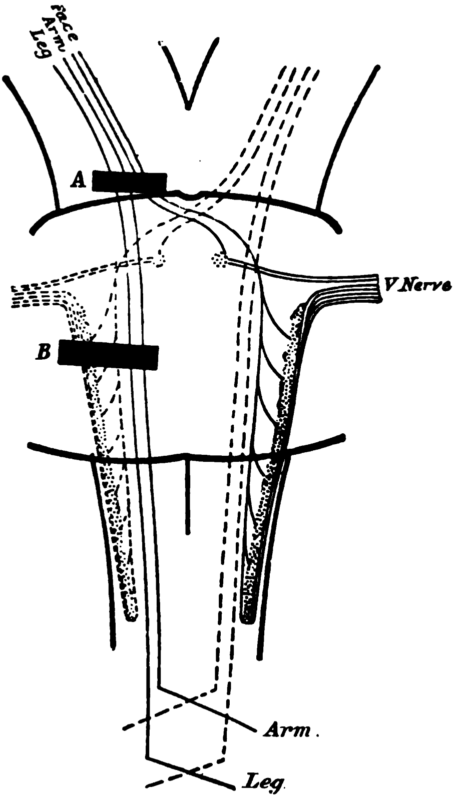

FIG. 10.—Course of Sensory Fibres in the Pons (Starr).

A. Lesion causing right hemianæsthesia.
B. Lesion causing "alternate hemianæsthesia" of left face and right side of body.

The sensory fibres from *muscles* and from *bones*, together with the uncrossed minority of tactile fibres, ascend uncrossed in the posterior column of the cord to the nucleus gracilis and nucleus cuneatus, the nucleus gracilis receiving the fibres from the lower limb, the nucleus cuneatus those from the upper limb. From these two nuclei, nerve fibres pass upwards, and cross the middle line in the internal arcuate fibres, forming the superior sensory decussation of the fillet (contrast this with the spinal decussation of the thermal, pain, and the majority of the tactile fibres). The sensory fibres from the leg, passing through the nucleus gracilis, cross lower down than those from the arm, which go through the nucleus cuneatus. Having crossed to the opposite side and reached the fillet, a flattened strand of fibres, they pass upwards in that tract not far from the thermal and pain fibres. The main mass of the tactile fibres ascends through the crus cerebri to the optic thalamus, and passes on through the internal capsule to the sensory cortex, which lies mainly behind the Rolandic fissure.[1]

We have still to consider another tract, which conducts sensory fibres for the *sense of equilibrium*. This tract does not commence in the posterior root-ganglion but arises as an "endogenous" tract within the cord. Arising from the cells of Clarke's column at the base of the posterior horn, it constitutes the direct cerebellar tract and ascends, uncrossed, into the restiform body and cerebellum.

Before leaving the motor and sensory tracts within the central nervous system it will be useful to mention the symptoms produced by a lesion of a lateral half of the spinal cord. Such a lesion is most commonly the result of a stab in the back; less commonly it is produced by bullet-wounds, fractured spine, or by chronic lesions of the spinal cord itself. The syndrome which results is known as **Brown-Séquard paralysis** and it will be readily understood by reference to Figs. 8 and 11. The symptoms are as follows:—

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[1] Sensory impulses run up not only to the post-central convolutions, but also to the motor areas. Thus a lesion of the motor cortex, besides causing a monoplegia of the corresponding limb, produces slight anæsthesia of the affected limb, with deficient sense of position in the weakened parts and diminished kinæsthetic sense in the affected muscles. It is to be borne in mind that the pyramidal motor cells do not lie in the most superficial layer of the cortex, but are covered by a layer of cells which are probably sensory in function. A striking evidence of this was afforded by a case of Negro and Oliva. These observers had the opportunity of stimulating the motor cortex of a woman who had been trephined. They found that with feeble stimulation only sensory symptoms were produced, a stronger stimulus being required to cause motor spasms.

(1.) *On the side of the lesion* we have, from interruption of the motor tract, motor paralysis of the corresponding leg, with an extensor plantar reflex from the outset and, later on, exaggeration of the deep reflexes. There is a slight elevation of temperature, owing to the interruption of vaso-motor fibres which descend in the lateral column. There is loss of sense of position on passive movement of the limb, due to interruption of the uncrossed fibres from the posterior roots below the lesion. The paralysed leg is not anæsthetic, but, just at the level of the lesion, there is around the trunk a narrow zone of anæsthesia to touch, temperature, and pain, from severance of a few sensory fibres cut across before they have succeeded in crossing to the other side. Finally, there is a narrow zone of hyperæsthesia above the anæsthetic zone, perhaps due to local irritation of the posterior-root fibres in the cord, close above the lesion; but this explanation is in dispute.

(2.) *On the side opposite to the lesion* there is no motor paralysis. But there is loss of cutaneous sensation to temperature and pain (completely), and to touch (partially), in the non-paralysed lower limb and in the corresponding half of the trunk up to the level of the lesion. The "vibration sense" (tested by a tuning-fork) in the bones of the leg on the uninjured side is also lost, since the path of the vibration-sense closely corresponds with that of the temperature sense. There is a zone of hyperæsthesia above the anæsthetic area, as on the side of the injury. Motor power is unimpaired, so also is the sensation of position on active or passive movement of the limb.

Cerebellum.—The cerebellum consists of a middle lobe or *vermis*, with a lateral lobe on each side, and its cortex is finely folded into leaves or lamellæ. Within the white matter, at a distance from the cortex, there are several important masses of grey matter, of which the chief are the corpora dentata, two hollow crumpled sacs, one within each lateral lobe; the roof nuclei within the middle lobe; and the nuclei of Deiters, or accessory vestibular nuclei, within the pons, one at each lateral aspect. Deiters' nucleus has several highly important connections, with the cerebellar cortex, with the ocular nuclei (third and sixth), and with the anterior cornual cells of the same side of the cord, through the vestibulo-spinal tract. This centre in Deiters' nucleus, with its various connections, is probably the lower reflex mechanism whereby, when we hear a sound, the head and eyes are promptly turned towards the side on which the auditory stimulus was received.

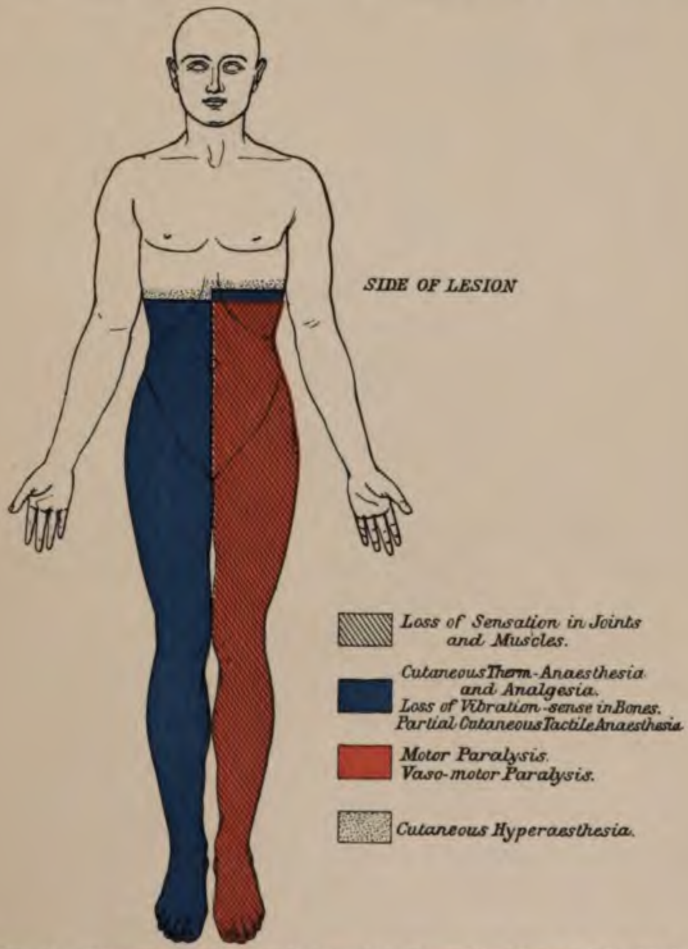
SIDE OF LESION
▨ Loss of Sensation in Joints and Muscles.
Cutaneous Therm-Anaesthesia and Analgesia.
Loss of Vibration-sense in Bones.
Partial Cutaneous Tactile Anaesthesia.
Motor Paralysis.
Vaso-motor Paralysis.
Cutaneous Hyperaesthesia.

FIG. 11.—Diagram illustrating the symptoms resulting from a left-sided hemi-section of the spinal cord (Brown-Séquard syndrome).

Each lateral lobe has three peduncles (Fig. 12). Of these, the superior peduncle contains fibres derived mainly from the dentate nucleus, this nucleus in turn being connected with the cerebellar cortex. The superior peduncle passes up ventral to the corpora quadrigemina, crosses the middle line and reaches the red nucleus in the crus cerebri of the opposite side. Starting again from this station, fibres run forwards along the anterior limb of the internal capsule, and pass to the optic thalamus and to the cortex of the frontal lobe. In this way the cerebellar hemisphere of one side is connected with the opposite side of the cerebrum, such connection being not a direct one, from cortex to cortex, but indirect, by the intermediation of intra-cerebellar and intra-cerebral nuclei.

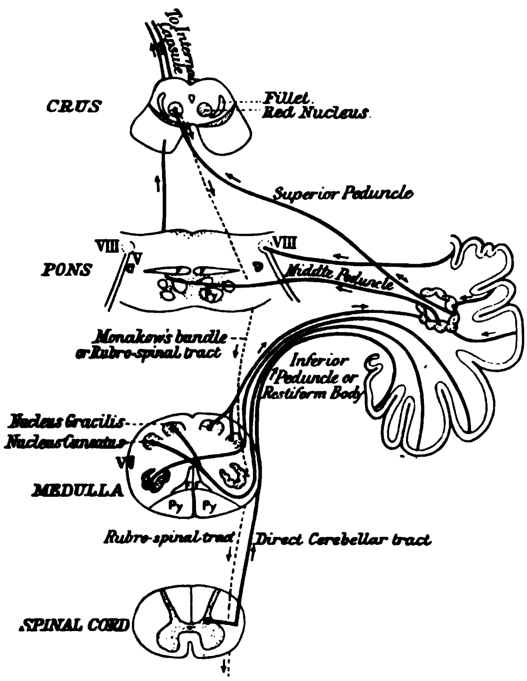

FIG. 12.—Diagram of chief connections of Cerebellum and of Red Nucleus.

The middle peduncle constitutes the greater part of the transverse fibres of the pons. And here again its fibres are not mere commissural strands running from one lateral lobe to the other.

Fibres from each lateral lobe cross the middle line, it is true, but they end in cells in the formatio reticularis of the opposite side of the pons. There new fibres arise and pass up the superior peduncle through the crus cerebri and internal capsule to the frontal, temporal, and occipital lobes of the cerebrum. Important fibres also connect Deiters' nucleus with the corresponding side of the cerebellar cortex in the region of the flocculus, conveying impulses to the cerebellum from the semicircular canals.

The inferior peduncle or restiform body connects the cerebellum with the medulla and spinal cord. It contains the direct cerebellar tract, conveying impulses of equilibration from the same side of the spinal cord, also arcuate fibres from the posterior column nuclei of both sides, and fibres from the inferior olive of the opposite side. All these fibres are afferent, leading upwards.

Broadly speaking, then, each lateral half of the cerebellum is in connection mainly with the cerebral hemisphere of the opposite or contra-lateral side, with both sides of the medulla, and with the same or homo-lateral side of the spinal cord.

Thus the cerebellum receives impulses from various sources :— from the cerebrum through the superior peduncles, from the skin and muscles through the inferior peduncles, and from most of the cranial nerve nuclei, especially from the semicircular canals, through the middle peduncles. And in turn the cerebellum sends efferent impulses to the cerebrum, reinforcing the general muscular tonus and co-ordinating the motor impulses proceeding from the cerebrum. In most voluntary movements the centre of gravity of the body is altered and certain muscular groups must co-ordinate to maintain the equilibrium. For this purpose the tonus of these muscular groups has to be augmented, and this is accomplished by the cerebellum, the great centre for co-ordination and equilibration, partly by its action on the cerebral motor cortex, partly, perhaps, through the nuclei of Deiters and the descending vestibulo-spinal tracts.

Experiments[1] have shown that definite motor centres exist in the cerebellar cortex. The right half of the vermis and the right cerebellar hemisphere contain motor centres for the right half of the body, and such centres can be identified by stimulation. If we stimulate the lateral parts of the vermis or the lateral cerebellar hemisphere, we produce movements of the homolateral limbs and also rotation of the body around its own longitudinal spinal axis. Thus excitation of the right side of the cerebellum produces a rotation in the direction of unscrewing an ordinary screw (the animal's head representing the head of the screw). Conversely, removal or division of the right lateral lobe produces rotation in the reverse direction—namely, that of screwing in a screw—since the intact side of the cerebellum maintains its tonic action, and is no longer opposed by the affected side whose muscles have lost their tonus. In such unilateral destructive lesions loss of co-ordinating influence also causes inco-ordination of the homolateral arm and leg, together with various ocular phenomena, including nystagmus, owing to loss of cerebellar influence on the ocular nuclei through Deiters' nucleus.

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[1] Pagano, *Rivista di patologia nervosa e mentale*, 1902, p. 145. Prus. *ref.* in *Neurologisches Centralblatt*, 1903, p. 268.

Stimulation of the vermis in the middle line produces bilateral movements. Thus, if the posterior end of the vermis be stimulated, the animal rotates violently forwards, whilst if the anterior part of the vermis be stimulated, the animal rotates violently backwards. Destruction of the middle lobe produces opposite effects, the animal tending to fall forwards from an anterior lesion of the vermis, and conversely.

The movements of the trunk and limbs which result from stimulation of the cerebellar cortex are probably not produced by direct action of the cerebellum on the spinal cord. Not only is there no direct tract leading downwards from the cerebellum into the cord but only an indirect path through Deiters' nucleus and the vestibulo-spinal tract, but Pagano has shown that if the contralateral cerebral cortex be extirpated, stimulation of the lateral lobe of the cerebellum no longer induces movements in the homolateral limbs. The motor action of the cerebellum is exercised indirectly, through the intermediation of the superior peduncles and the cerebral cortex.

LECTURE II

ANATOMY AND PHYSIOLOGY (*continued*)

Having considered the chief motor and sensory tracts within the central nervous system, let us now pass to the peripheral paths whereby the central nervous system is connected with the various end-organs.

Firstly, as to the motor system. Motor fibres starting in the cortex, and passing down the pyramidal tract, as already described, reach the nuclei of the various motor cranial nerves in the crura, pons, and medulla. Then, passing along the spinal cord, the pyramidal tract gives off fibres to the anterior cornua at various levels all the way down. This portion of the motor tract, from the motor cortex to the extremity of the pyramidal fibres, is called the *upper motor neurone*, and if the cortical motor cell or its axon, the pyramidal motor fibre, be destroyed, we have degeneration of the whole length of the pyramidal fibre below the level of the lesion, stopping short when it reaches the anterior cornual cell. This so-called "descending degeneration" does not begin at the lesion and spread downwards as the name might suggest, but affects simultaneously the whole motor fibre below the lesion, on the side remote from the nerve-cell which is its trophic centre.

From each anterior cornual cell a new motor fibre passes out of the cord along an anterior nerve-root, enters into the formation of a peripheral motor nerve, and thereby is conducted to a muscle-fibre. This lower segment of the motor path, starting at the anterior cornual cell and ending in the muscle-fibre, is called the *lower motor neurone*. Here also, if the anterior cornual cell or its axon, the peripheral motor nerve, be destroyed, we again have a "descending degeneration" of the whole fibre on the distal side of the point of injury, and of the muscle-fibre also. We note that degeneration of the upper motor neurone does not spread into the lower neurone, nor *vice versâ*. It is particularly to be remembered that a lesion of the upper motor neurone leaves the lower reflex arc intact, whilst a lesion of the lower motor neurone not only severs the reflex arc, but also causes the muscle-fibre in that reflex arc to degenerate and waste away.

Therefore in diagnosing the position of the lesion, in any given case of motor paralysis due to organic disease, the first question me must ask ourselves is whether the lesion is in the upper (cortico-spinal) or in the lower (spino-muscular) motor neurone. There is usually little difficulty in answering this question, if we bear the following points in mind :—

Lesion of Upper (Cortico-Spinal) Motor Neurone.	**Lesion of Lower (Spino-muscular) Motor Neurone.**
1. Motor Paralysis.	1. Motor Paralysis.
2. Spasticity.	2. Flaccidity.
3. No muscular wasting (apart from disuse).	3. Muscular Atrophy.
4. Electrical reactions normal.	4. R.D. (reactions of degeneration).
5. Deep reflexes present and often increased.	5. Deep reflexes absent or diminished.
6. Extensor plantar reflex (if foot affected).	6. Plantar reflex, if present, of normal flexor type (unless lesion paralyses flexor muscles themselves).

Having thus recognised which motor neurone, upper or lower, is affected, we have then to decide at what level in the affected neurone the lesion is situated. To that point we shall come later (p. 181).

Returning for a moment to the sensory fibres; these may also be regarded as arranged in *sensory neurones*. The lowest sensory neurone starts from a sensory end-organ, in the skin or elsewhere, and extends up to the nerve-cell in the intervertebral ganglion on the posterior spinal root. This ganglion-cell is the trophic centre for the peripheral sensory fibre, and a lesion at or below this cell will cause "descending degeneration" of the distal segment of the fibre and of the peripheral end-organ. There is, however, one sensory end-organ, the muscle-spindle, which is an exception to this rule, and which does not degenerate when the afferent fibre leading from it to the ganglion-cell is destroyed. The muscle-spindle thus has its trophic centre within itself.

But the ganglion-cell of the posterior root is also the trophic centre for the fibre which passes upwards from it along the pos-

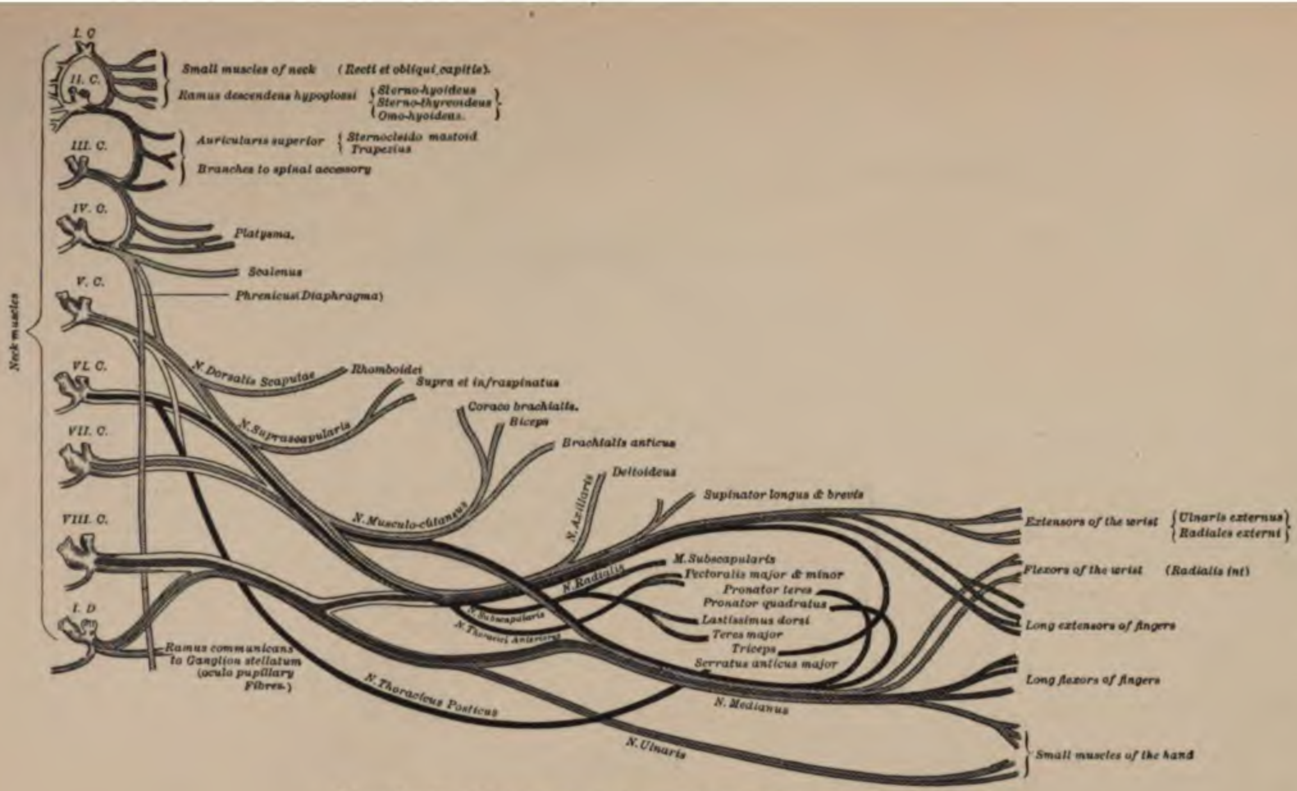


FIG. 13.—The Cervico-brachial Plexus and its Branches (Kocher).

terior root and enters the posterior column of the spinal cord. And therefore a lesion at or above the intervertebral ganglion-cell, in the posterior root, or in its intra-spinal prolongation in the posterior column, will cause an "ascending degeneration" of the fibre through its whole course within the spinal cord. Here again this so-called "ascending" degeneration occurs simultaneously throughout the entire extent of the nerve-fibre, on the side remote from its trophic centre in the intervertebral ganglion; and this degeneration extends as far as, but does not implicate, the next nerve-cell whose

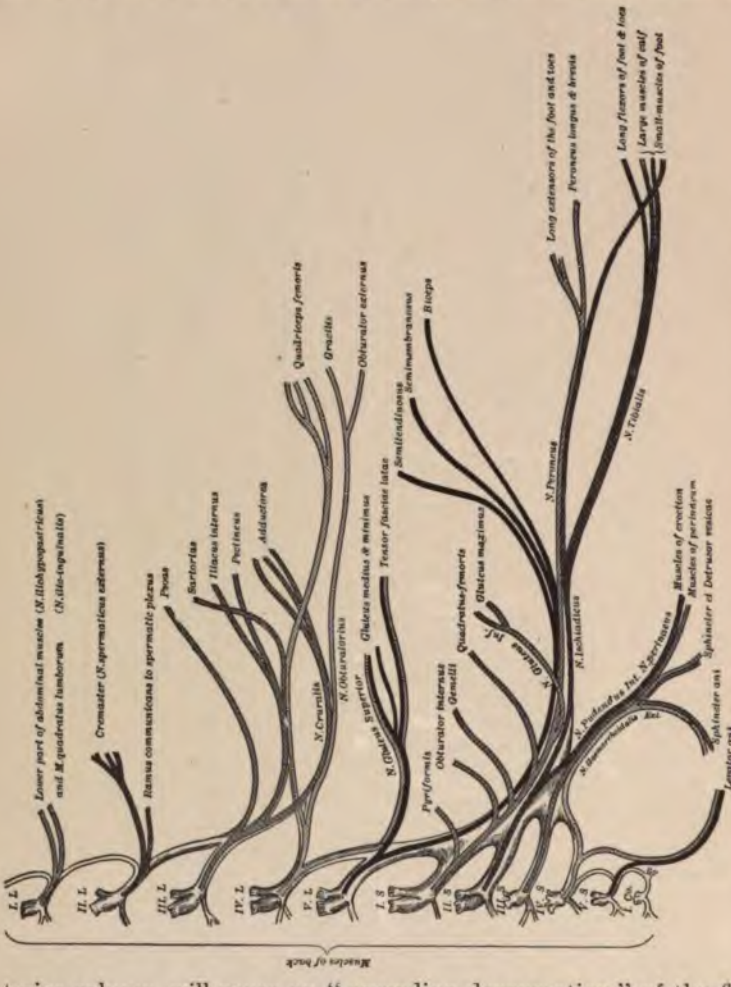


FIG. 14.—The Lumbo-sacral Plexus and its Branches (Kocher).

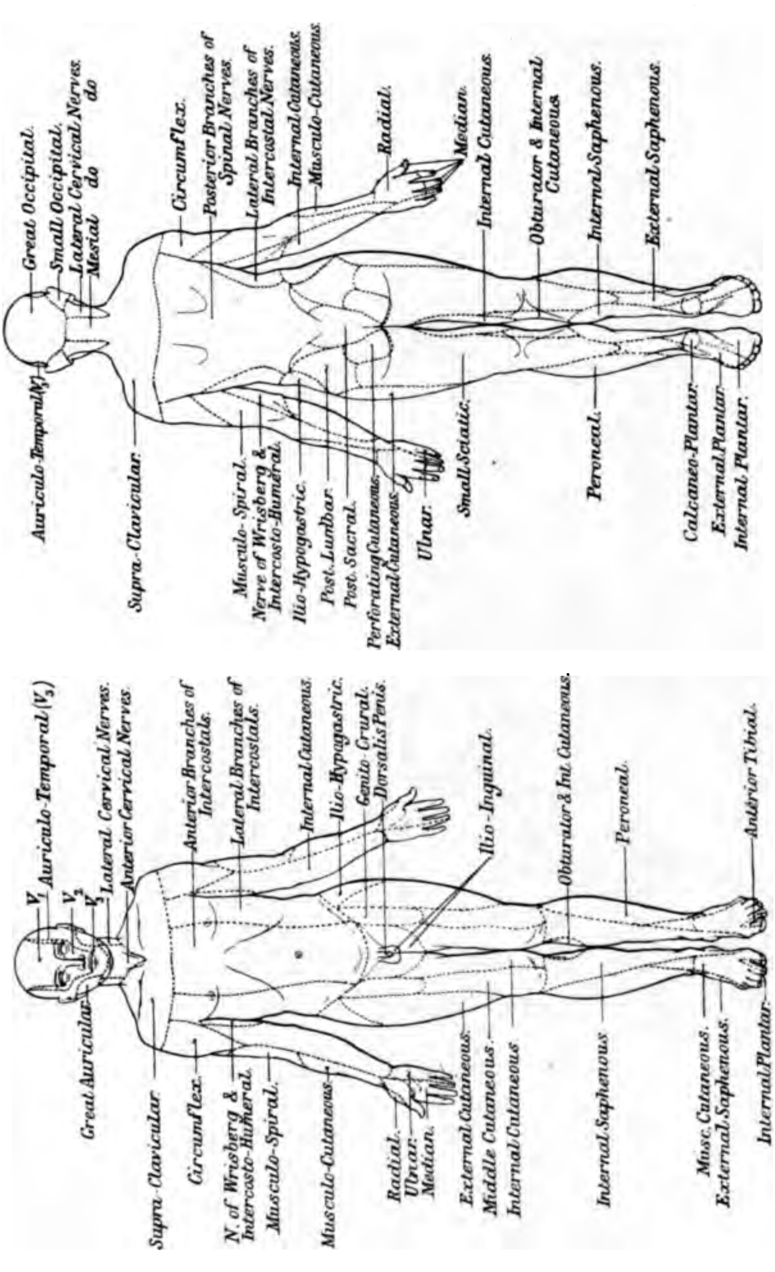
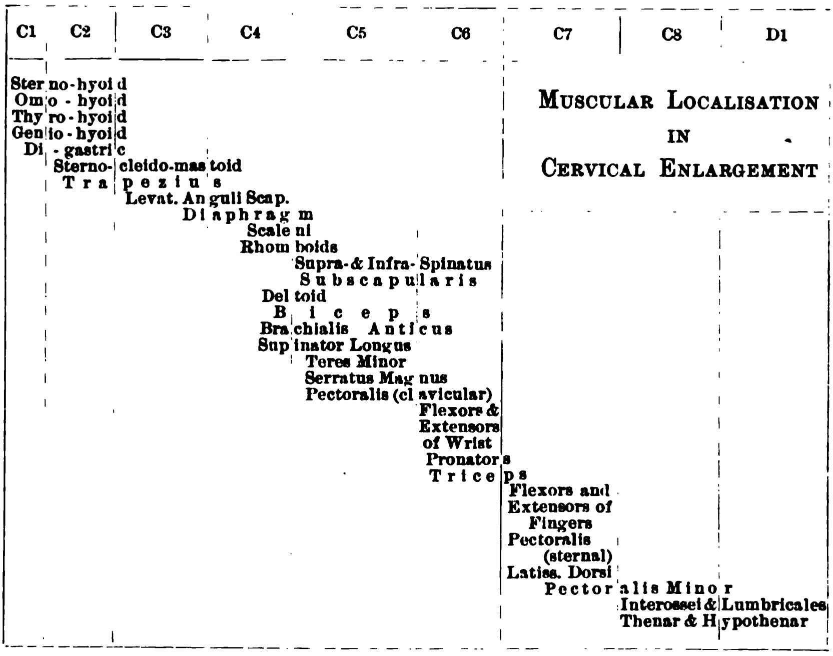


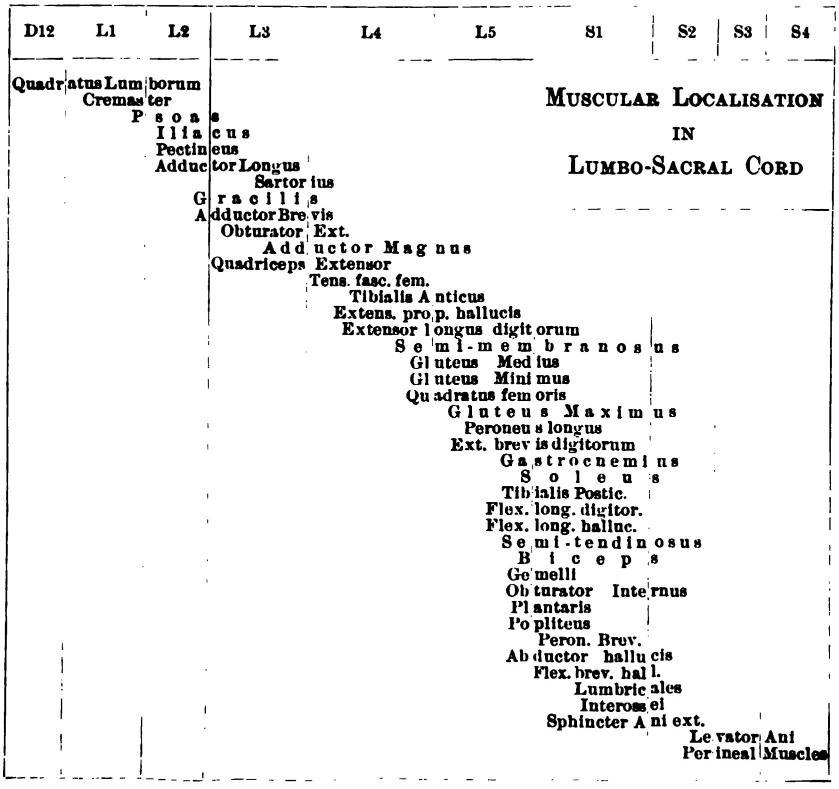
FIG. 15.—Diagram of Cutaneous Areas of Peripheral Nerves.

axon leads upwards towards the brain. Similarly a lesion of this second sensory neurone causes ascending degeneration in the fibre of the fillet above, as far as, but not including, the optic thalamus. There a third neurone starts, leading up to the sensory area of the cerebral cortex. In the case of the ordinary sensory tract, the path traverses the internal capsule. In the case of an impulse which traverses the cerebellum, the chain of successive neurones is more complex, and consists, firstly of a posterior-root neurone, secondly of an ascending spino-cerebellar neurone, thirdly of a cerebello-dentate, fourthly of a dentato-thalamic, and lastly of a thalamo-cortical neurone. The so-called "Wallerian degeneration" —"ascending" or "descending" as the case may be—signifies that a nerve-fibre, separated from its trophic cell, degenerates on the side remote from that cell.

The anterior and posterior nerve-roots join to form mixed nerve-trunks. These again branch and intermingle to form plexuses in the cervical, lumbar, and sacral regions. The distribution of the different nerves, motor and sensory, is represented in Figs. 13, 14, and 15.

Root Lesions.—But what is perhaps less familiar, though of equal diagnostic importance, is the distribution of the anterior and posterior spinal roots. In lesions of the lower motor neurone, we have often to decide whether the distribution of symptoms points to a lesion of a peripheral nerve-trunk, such as the musculo-spiral or sciatic, or whether it points to a lesion of one or more nerve-roots before they have joined to form the trunks of a plexus. Thus, for example, the deltoid is frequently paralysed alone owing to a lesion of the circumflex nerve, but it is never paralysed alone as a result of a lesion of the anterior cornu or anterior nerve-root. Again, a lesion of the musculo-spiral nerve may produce paralysis both of the supinator longus and of the extensors of the wrist and fingers, but these muscles are never affected together by a lesion of a single segment of the spinal cord or of a single anterior root, since their motor cells lie at different levels in the anterior cornu. This will be readily seen on studying the following tables, which show the nuclear representation of muscles in the anterior cornua at various levels of the spinal cord.


C1
C2
C3
C4
C5
C6
C7
C8
D1
MUSCULAR LOCALISATION IN CERVICAL ENLARGEMENT
Sterno-hyoid
Omo-hyoid
Thyro-hyoid
Genio-hyoid
Di-gastric
Sterno-cleido-mastoid
Trapezius
Levat. Anguli Scap.
Diaphragm
Scaleni
Rhomboids
Supra- & Infra-Spinatus
Subscapularis
Deltoid
Biceps
Brachialis Anticus
Supinator Longus
Teres Minor
Serratus Magnus
Pectoralis (clavicular)
Flexors & Extensors of Wrist
Pronators
Triceps
Flexors and Extensors of Fingers
Pectoralis (sternal)
Latiss. Dorsi
Pectoralis Minor
Interossei & Lumbricales
Thenar & Hypothenar


D12
L1
L2
L3
L4
L5
S1
S2
S3
S4
MUSCULAR LOCALISATION IN LUMBO-SACRAL CORD
Quadratus Lumborum
Cremaster
Psoas
Iliacus
Pectineus
Adductor Longus
Sartorius
Gracilis
Adductor Brevis
Obturator Ext.
Adductor Magnus
Quadriceps Extensor
Tens. fasc. fem.
Tibialis Anticus
Extens. prop. hallucis
Extensor longus digitorum
Semi-membranosus
Gluteus Medius
Gluteus Minimus
Quadratus femoris
Gluteus Maximus
Peroneus longus
Ext. brevis digitorum
Gastrocnemius
Soleus
Tibialis Postic.
Flex. long. digitor.
Flex. long. halluc.
Semi-tendinosus
Biceps
Gemelli
Obturator Internus
Plantaris
Popliteus
Peron. Brev.
Abductor hallucis
Flex. brev. hall.
Lumbricales
Interossei
Sphincter Ani ext.
Levator Ani
Perineal Muscles

We observe that comparatively few muscles are confined to a single segment, but that most of them are represented in two or more segments. If, therefore, a cord lesion be limited to one segment, it will cause complete paralysis of the muscles confined to that particular segment and partial paralysis of muscles whose motor nuclei extend up or down into other segments. This explains the apparent irregularity in the distribution and degree of paralysis in certain cases of infantile paralysis.

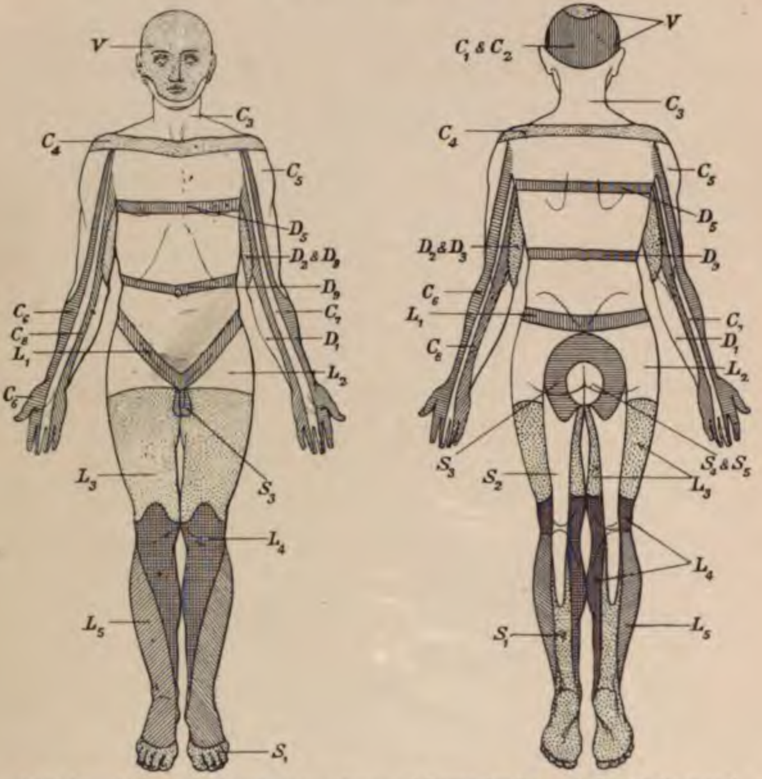


FIG. 16.—Diagram of Cutaneous Areas of Posterior Roots (after Collier and Purves Stewart).

Similarly in a case of cutaneous anæsthesia it is important to distinguish between a posterior-root lesion and a lesion of a peripheral nerve-trunk such as the radial or ulnar. The distribution of the posterior nerve-roots is indicated diagrammatically in Fig. 16.

Paths of Special Senses.—**Olfactory Path.**—The olfactory nerves, about twenty on each side, arising from the under surface of each olfactory bulb, perforate the cribriform plate of the ethmoid bone and are distributed to the olfactory region of the nasal mucous membrane. The central olfactory tract has various connections, not of great clinical importance, leading to the optic thalamus and to other sub-cortical ganglia. It connects the olfactory bulb with the cortical centre for smell, situated, as we have already seen, in the uncinate gyrus at the tip of the temporo-sphenoidal lobe (Fig. 4). The olfactory tract does not traverse the internal capsule. Each olfactory bulb is connected not only with the uncinate gyrus of the same side, but also, through the anterior commissure, with that of the opposite side.

Visual Path.—This is of great clinical importance. Starting from the retina, the visual fibres run backwards along the optic nerve. At the optic chiasma there is a partial decussation, so that the fibres from the left halves of both retinae (corresponding to the right halves of the visual fields) run together in the left optic tract, and *vice versâ*. The central visual impulses, from the macula lutea, pass into both optic tracts. The fibres of each optic tract run backwards, winding around below and to the outer side of the crus cerebri, to the primary optic centres, viz. :—the posterior part of the optic thalamus, the external geniculate body, and the superior corpus quadrigeminum.

From these three stations new fibres arise, forming the " optic radiation," passing through the internal capsule behind the fibres for common sensation (Fig. 6) and so reaching the cortical half-vision centre. This centre, mainly on the mesial aspect of the hemisphere, is in the cuneate lobe, which is divided into an upper and a lower part by the calcarine fissure (Fig. 4). The half-vision centre also extends on to the convexity of the occipital lobe (Fig. 3). The calcarine fissure forms a boundary-line between the cortical representations of the upper and lower quadrants of the corresponding half of the visual field. Therefore whilst a lesion of the left occipital lobe, or of the whole of the left cuneus, or of the fibres of the left optic radiation, will cause a right-sided hemianopia in both visual fields, a lesion of the left cuneus, if limited to the part above the calcarine fissure, will cause blindness of the right lower quadrant of both visual fields, whilst if the lesion of the cuneus be below the left calcarine fissure it will produce blindness of the right upper quadrant of both fields. These are varieties of " quadrantic hemianopia."

Besides the half-vision centre, there is a higher centre on the convex surface of the occipital cortex, where a lesion, if sufficiently superficial (so as to miss the subjacent optic radiation), may cause, not hemianopia, but what is called " crossed amblyopia." This means a concentric contraction of both visual fields, most marked in the eye of the side opposite to the side of the lesion. Such a lesion has not been conclusively demonstrated in gross organic disease, but crossed amblyopia is one of the most frequent symptoms in hysteria.

Finally, in right-handed people there is in the left angular gyrus a centre for the recognition of visual memories of written and printed speech. If this centre be destroyed, we have word-blindness, which may or may not be associated with right hemianopia, according as the subjacent optic radiations are affected or not. Fig. 17 indicates diagrammatically these various fibres and centres, and also shows the effects upon the visual fields of lesions in various parts of the visual path.

Gustatory Path.—The course of the taste-fibres outside the brain is somewhat complex, and we shall study it again later when we consider the cranial nerves. It is probable that many of the taste impulses enter the brain through the sensory root of the fifth cranial nerve, a smaller number through the glosso-pharyngeal nerve, and some through the *nervus intermedius* of Wrisberg. The cortical centre for taste is probably in the front part of the temporo-sphenoidal lobe, close to the olfactory centre (Fig. 4). The intra-cerebral course of the gustatory fibres is not definitely settled, but it is probable that they do not traverse the internal capsule.

Auditory Path.—This is of some practical importance. Entering the medulla in the cochlear division of the eighth nerve, the auditory fibres embrace the restiform body, some passing along its inner side to the ventral auditory nucleus, others passing along its outer side to the dorsal auditory nucleus. From these two nuclei new fibres pass upwards towards the cortex. A few run up uncrossed in the fillet of the same side, but most of the fibres decussate and ascend in the fillet of the opposite side. Some end in the posterior corpus quadrigeminum, others go on to the corpus geniculatum externum, and finally the bulk of them, passing through the sub-lenticular region of the internal capsule behind the sensory fibres, reach the cortical auditory centre in the first temporo-sphenoidal convolution (Fig. 3). We note that each cortical centre receives auditory messages from both ears, though more extensively from the ear of the opposite side, and that therefore a lesion limited to one temporal lobe will not cause deafness. In right-handed people there is a specially differentiated portion of the left auditory centre where memories of word-sounds are stored up (Fig. 3).

A certain amount of clinical evidence[1] supports the view that there is a special cortical centre associated with the sensations of *hunger* and *thirst*. It would appear to be in the temporo-sphenoidal lobe, at or near the olfactory centre. Abscesses, injuries and tumours in this region have sometimes been associated with voracious appetite and intense thirst, persisting for weeks or months.

Arteries of the Brain.—Most cases of acute brain disease which we meet with in practice are the direct result of some vascular disease, *e.g.* hæmorrhage, thrombosis, or embolism. It is therefore important to understand certain facts about the cerebral circulation.

The brain is supplied by two pairs of arteries—the internal carotids and the vertebrals, as shown in Fig. 18. The two vertebral arteries join to form the basilar artery which runs forwards in the middle line along the front of the pons, supplying perforating branches to the pons and arteries to the cerebellum. Between the crura cerebri the basilar divides into the two posterior cerebral arteries, each of which winds round the outer side of the crus, supplying it as it passes, and also giving branches to the optic thalamus and the corpora quadrigemina. Finally it reaches and supplies the lower part of the temporo-occipital cortex (Fig. 19). Each posterior cerebral

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[1] Stephen Paget, *Essays for Students*, 1898.

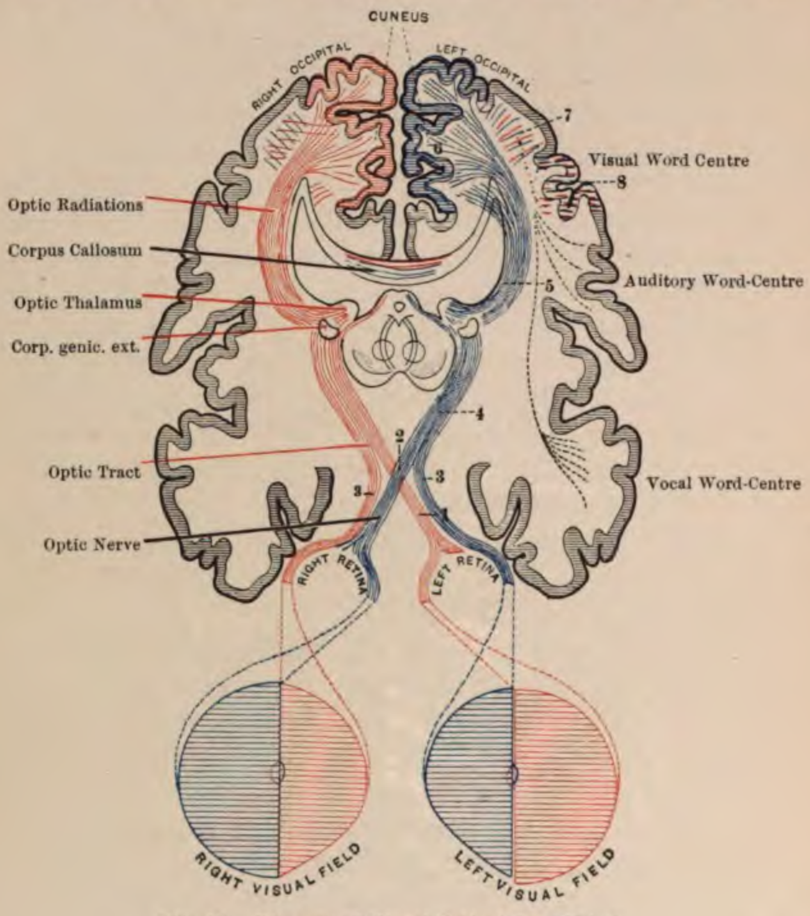

FIG. 17.—Diagram of Visual Paths (after Vialet).

Lesion at 1	produces	Blindness of one eye.
,, at 2	,,	Bi-temporal Hemianopia.
,, ,, 3 and 3	,,	Bi-nasal Hemianopia.
,, ,, 4	,,	R. Hemianopia with Hemiopic Pupil Reaction.
,, ,, 5	,,	,, with Normal Pupil Reaction.
,, ,, 6	,,	,, ,, ,, ,,
,, ,, 7	,,	Crossed Amblyopia.
,, ,, 8	,,	Word-Blindness.

artery sends a posterior communicating artery forwards to join the internal carotid.

The internal carotid divides into three branches:—anterior cerebral, middle cerebral, and posterior communicating. The two anterior cerebral arteries are connected by the short anterior communicating artery, thus completing the "circle of Willis." The anterior cerebral artery passes forward, and then, curving round

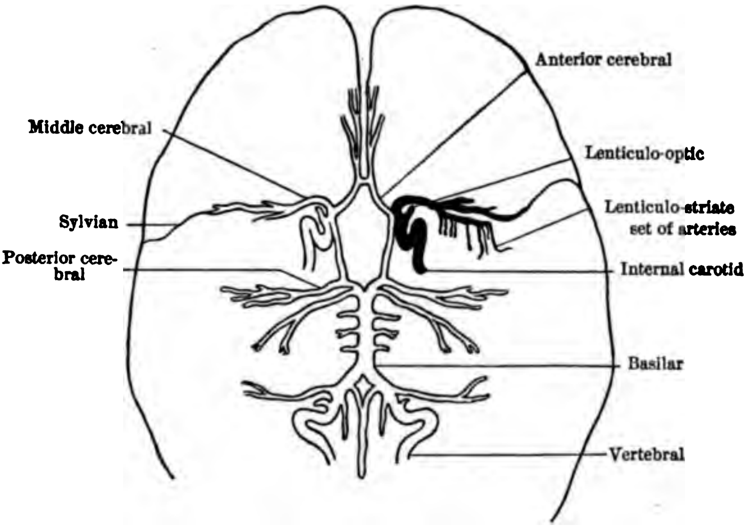

FIG. 18.—Arteries at the Base of the Brain. One, the lenticulo-striate, is called the artery of cerebral hæmorrhage. (After Dercum.)

to the top of the corpus callosum, it turns backwards, parallel with its fellow of the opposite side, between the mesial surfaces of the two hemispheres. Most of the mesial surface of the cerebral hemisphere, as far back as the parieto-occipital fissure, is supplied by the anterior cerebral artery (Fig. 19). It also sends branches over the edge of the hemisphere to its convex surface, supplying the anterior part of the frontal convolutions. And at the beginning of its course, it sends a few perforating branches inwards to the caudate nucleus.

The middle cerebral artery is clinically the most important of the three. Its main trunk passes upwards and outwards along the Sylvian fissure to the surface of the island of Reil where it divides into its terminal branches. At its beginning it gives off numerous basal perforating arteries, which enter the anterior perforated space and ascend to the corpus striatum. These are called lenticular, lenticulo-striate, and lenticulo-optic, according to their distribution. All these, and especially one of the lenticulo-striate arteries, are frequently the seat of cerebral haemorrhage. The main trunk of the middle cerebral runs along the Sylvian fissure, where it divides into four terminal branches (Fig. 19). One goes to Broca's convolution (the third inferior frontal); another to the lower two-thirds of the ascending frontal convolution and to the adjacent part of the second frontal convolution; another to the ascending parietal convolution of the adjacent intra-parietal lobule; and a fourth to the supra-marginal convolution, the angular gyrus and the upper temporo-sphenoidal convolutions, and so to the tip of the lobe on its mesial aspect.

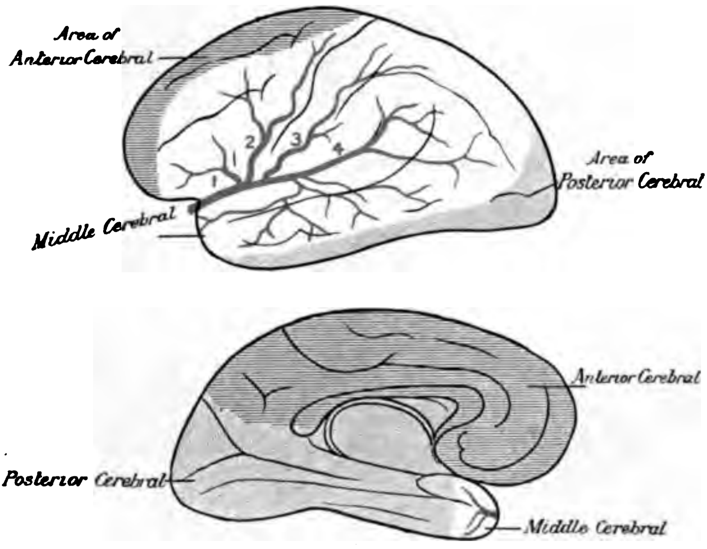

FIG. 19.—Diagram of arterial supply of cortex.

The cortical arteries anastomose freely with one another; but not so the perforating basal vessels. They are "terminal" arteries, and do not anastomose either with each other or with the cortical vessels. Therefore if a basal artery becomes blocked by thrombosis or embolism, a permanent area of necrosis results. On the other hand, the blocking of a cortical artery admits of a more favourable prognosis, since a collateral circulation may develope and the necrotic process be arrested.

The *cerebellum* is supplied by the anterior cerebellar and superior cerebellar arteries from the basilar, and by the posterior cerebellar artery from the vertebral.

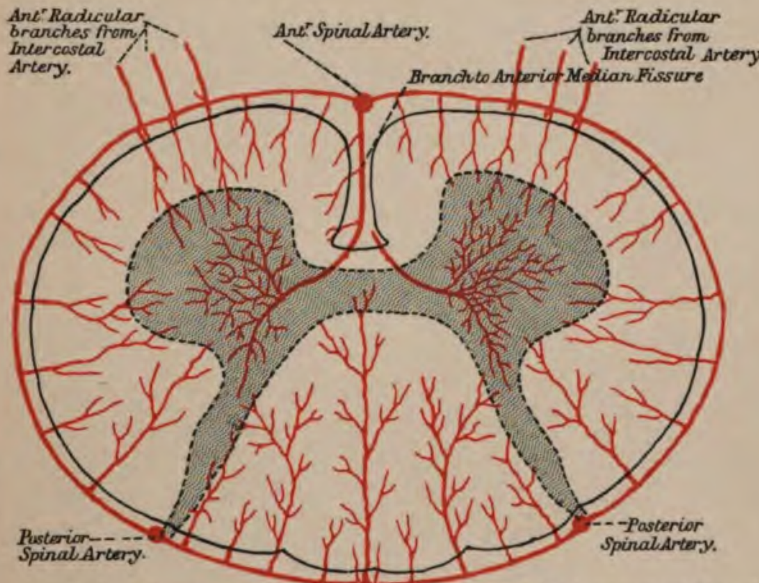
Antʳ Radicular branches from Intercostal Artery.
Antʳ Spinal Artery.
Antʳ Radicular branches from Intercostal Artery.
Branch to Anterior Median Fissure
Posterior Spinal Artery.
Posterior Spinal Artery.

FIG. 20.—Diagram of the course and distribution of the terminal arteries of the spinal cord. (After Van Gehuchten.)

The *spinal cord* is supplied by three main arteries, one anterior and two posterior, running on the surface along the entire extent of the cord. The anterior spinal artery arises from one or other vertebral, receiving a small communicating branch from the opposite vertebral. As it runs down the front of the cord, it is reinforced by a series of smaller vessels, derived from the intercostal and lumbar arteries, which enter here and there along the anterior roots. The anterior spinal artery sends numerous branches deeply along the pia mater lining the median fissure, dipping alternately into the right and left sides of the fissure, and supplying the grey matter of the cord. The two posterior spinal arteries, one on each side, also arise from the vertebral arteries and run downwards on the back of the cord, close to the posterior roots, being reinforced by small branches entering here and there along the posterior roots. Fig. 20 shows diagrammatically the position of these various spinal arteries, and it should be observed that the grey matter and the white receive their blood-supply from different vessels. Moreover, like the perforating basal arteries of the brain, all the spinal arteries, once they have penetrated the cord, are terminal arteries and do not anastomose. Therefore embolism or thrombosis of a spinal artery always produces an area of necrosis.

The **Venous Circulation** in the brain is peculiar, inasmuch as the direction of the blood-stream in the cortex is the same in the arteries as in the veins, *i.e.* from before backwards. The superior longitudinal sinus receives not only the superior cerebral veins from the cortex, but also some veins from the scalp, and through its starting-point at the foramen cæcum it receives branches from the nasal vein, though after childhood this foramen often becomes closed. Therefore when *thrombosis of the superior longitudinal sinus* occurs in a marasmic child, we expect epistaxis and distension of the nasal veins, together with distension of the veins of the scalp. Posteriorly the superior longitudinal sinus ends at the torcular Herophili.

The inferior longitudinal sinus is quite small, and, like the superior, lies between the layers of the falx cerebri, but at its lower edge. It runs backwards to the anterior edge of the tentorium, receiving branches from the mesial surface of the hemispheres, and ends in the straight sinus. The straight sinus receives some cerebellar veins and the veins of Galen from the velum interpositum and interior of the brain, and passes backwards either into the torcular Herophili or into one of the lateral sinuses. When the veins of Galen are thrombosed the cerebral ventricles become distended with fluid.

The lateral sinuses begin at the internal occipital protuberance and arch outwards, one on each side, to open through the jugular foramen into the internal jugular vein. The sinus passes close to the mastoid portion of the temporal bone. Here it receives the superior petrosal sinus, and also emissary veins from the scalp in the mastoid region. Just before it empties into the jugular, it receives the occipital sinus from the torcular. *Lateral sinus thrombosis* is a well-known and dangerous complication of suppuration in the middle ear. It is recognised by distension of the mastoid veins, œdema of that region, and hardness and tenderness of the internal jugular vein.

The cavernous sinuses lie one on each side between the sphenoidal fissure and the apex of the temporal bone. Each sinus receives the ophthalmic veins from the orbit and communicates by means of the circular sinus with its fellow of the opposite side. The sinus ends posteriorly by opening into the two petrosal sinuses. *Thrombosis of the cavernous sinus* is generally secondary to some septic condition of the orbit or naso-pharynx. It is recognised by the presence of chemosis (œdema of the conjunctiva), proptosis (bulging forwards of the eyeball), and œdema of the upper lid and root of the nose. There may be paralysis of certain of the external ocular muscles from affection of the third, fourth, or sixth nerves which lie in the outer wall of the cavernous sinus.

Nearly all the intra-cranial venous blood leaves the skull by the internal jugular veins, so that in an infective thrombosis of any of the cerebral sinuses it may therefore become necessary to ligature the internal jugular vein, to prevent a general pyæmia.

LECTURE III

METHOD OF CASE-TAKING

In no class of maladies is a thorough examination of the patient of greater importance than in cases of nervous disease. One cannot urge too strongly the value of systematic examination, though it matters little what particular scheme of case-taking be adopted, so long as it is one which ensures that the investigation is complete, so that no point of importance is overlooked. Sometimes, it is true, we may make a diagnosis at a glance, as for example in paralysis agitans, or chorea. But more usually the patient presents symptoms or signs which are common to several diseases, and we have to decide which of these diseases he is suffering from. The chief causes of wrong diagnosis are insufficient examination, inaccurate observation, and, less commonly, false conclusions from correct and sufficient facts. But if we pursue a good routine method of examination, gross errors of diagnosis can generally be avoided.

Examination of a nervous case should not be confined to the nervous system alone. All the systems of the body should be investigated. An accomplished neurologist must be in the first place a sound physician.

The value of negative as well as positive facts cannot be over-estimated. The neurological student should accustom himself from the outset not only to chronicle deviations from the normal, but also, if he finds that certain other functions are normal, to record the fact and not to pass them over without reference.

As in any other case, a careful history should first be obtained. It is comparatively seldom that a patient, however willing he may be, provides us spontaneously with an accurate history of his case. We generally have to guide his tale by suitable questions, and in many cases we have to supplement the patient's account by inquiring for corroborative or correcting facts from the patient's friends.

We first inquire as to the *mode of onset* of the patient's symptoms, whether such onset was sudden or gradual, and, if gradual, the exact order in which the various symptoms appeared. In the *family history*, inquiry should be made as to the occurrence of nervous or mental disease in other members of the family, also as to a family history of gout, tuberculosis, cancer, &c. In certain cases, consanguinity of the parents should be inquired for. In the account of the patient's *previous health*, it is important to inquire as to syphilis, kidney disease, gout, rheumatic fever, or any previous nervous disease, and a note should be made as to the patient's occupation and habits of life, and as to his temperance in alcohol, tobacco, &c. When inquiring about venereal disease, if the patient is a man, we should put our questions straightforwardly, but in female patients considerable delicacy is necessary, and it is advisable simply to inquire for collateral symptoms, *e.g.*, rashes, falling out of hair, sore throat and, especially in married women, a succession of premature or dead children. In any case we should never interrogate a male patient on the subject of venereal disease in the presence of his wife, or *vice versâ*.

Having thus noted the chief points in the history of the case, we proceed to the examination of the patient's *present state*. The following scheme will be found useful :—

Scheme for Routine Examination of Nervous System.

Higher Cerebral and Mental Functions.

Intellectual functions—Emotional state—Memory—Hallucinations or delusions—Delirium—Coma—Drowsiness—Insomnia.

Fits or other Abnormal Movements.

Fits — Tremors — Fibrillary movements — Chorea — Athetosis — Tic — Myoclonus, &c. Description of each.

Speech and Articulation.

Aphasia—Is patient right or left handed ?

Cranial Nerves.

1. Smell—Anosmia—Parosmia.
2. Visual acuity—Fields of vision : Hemianopia, &c.—Colour-blindness —Ophthalmoscopic examination—optic discs, atrophy, neuritis—retinal hæmorrhages, choroiditis, &c.

3.
4.
6. } Pupils: Size, shape, reaction to light (direct and consensual), and to accommodation—External ocular movements—Ptosis—Movements of eyes in all directions—Convergence—Squint—Diplopia —Nystagmus.

5. Sensation—Face: Conjunctival, nasal, and buccal mucous membranes —Taste.

Motor—masseters, temporals, pterygoids, &c.

7. Facial muscles, upper and lower—Chorda tympani: taste in anterior two-thirds of tongue—Nerve to stapedius—hyperacousis?

8. Hearing: Aerial and bone conduction—Examination of meatus and tympanic membrane—Tinnitus—Vertigo.

9. Taste: Posterior third of tongue—Anæsthesia of pharynx—Difficulty in swallowing.

10. Palate—Recurrent laryngeal branch—Laryngoscopic examination—Heart, respiration, digestion?

11. Sterno-mastoid and Trapezius.

12. Tongue (motor only).

Sensory Functions.

Subjective sensations: Pain:—site, character, frequency—Headache—Vertigo—Tingling—"Pins and Needles"—Formication, &c.

Sensibility to touch—pain—temperature—Localisation of the spot touched—Vibration-sense (with tuning-fork)—Anæsthesia—Paræsthesia—Hyperæsthesia—Tenderness on pressure over nerve-trunks, muscles, or skin—Joint-sense—Sense of active muscular contraction with different weights—Astereognosis.

Motor Functions.

Motor Paralysis or Paresis:—In head and neck, upper limbs, diaphragm, intercostals, spinal and abdominal muscles, lower limbs.

Monoplegia—Hemiplegia—Diplegia—Paraplegia—Hemi-paraplegia—"Crossed paralysis," &c.

Co-ordination:—Unsteadiness of upper or lower limbs on voluntary movement—Gait.

Muscular Atrophy or Hypertrophy: Rigidity—Flaccidity—Hypotonia.

Reflexes.

Superficial.—Conjunctival, palatal, epigastric, abdominal, cremasteric, plantar (flexion or extension of hallux)—bulbo-cavernosus—anal.

Deep.—Jaw, wrist, elbow, knee, ankle-jerks. Ankle-clonus—knee-clonus, &c.

Organic.—Micturition—Retention—Retention with overflow incontinence —Intermittent incontinence.—Constant paralytic dribbling—Defæcation—Control of sphincters—Priapism.

Trophic Functions.

Muscles.—Electrical reactions—Faradic—Galvanic.

Skin.—Bullæ—Herpes—Bed-sores—Perforating ulcers—Glossy skin, &c.

Joints and Bones.—Charcot's arthropathy—Spontaneous fractures—Pes cavus, &c.

Examination of Skull and Vertebral Column.

Abnormal projections or depressions—Tenderness, &c., on percussion.

Cerebro-spinal Fluid.

Naked-eye characters of fluid obtained by lumbar puncture—Microscopic examination of centrifuged deposit—leucocytosis, &c.—Bacteriological characters—Chemical reactions, &c.

Sympathetic Nervous System.

Cervical sympathetic—Dilatation of pupil to shade and cocaine—Ciliospinal reflex — Proptosis — Exophthalmos — Enophthalmos—Retraction of upper lid—Pseudo-ptosis—Flushing or sweating of face, neck, upper extremity.

Angio-neuroses—Raynaud's disease—Erythromelalgia—Angio-neurotic œdema—Localised hyperidrosis or anidrosis—Intermittent limp, &c. &c.

Several points are worthy of notice in the general outline of the foregoing scheme. We begin with the higher cerebral and mental functions, for this reason, that if a patient be mentally obtuse, or worse, if he be delirious, then any statement he may make must be open to doubt, and for our diagnosis we must rely mainly, and in cases of coma entirely, upon physical signs and upon the history supplied by the patient's friends.

The patient's emotional state is sometimes of diagnostic significance. Not only are many hysterical patients unduly emotional, but a similar condition is observed in certain cases of disseminated sclerosis, where there is a tendency to smile and giggle upon slight provocation, whilst, on the other hand, cases of aphasia and of advanced bulbar palsy are often lachrymose.

The statements of a patient who is addicted to alcoholic intemperance or to chronic poisoning with opium, cocaine or other drug, must also be received with considerable scepticism. There is a special variety of loss of memory, called *Korsakow's psychosis*, which occurs in chronic alcoholics, where the patient, who is usually a woman and the subject of chronic alcoholic neuritis, has a faulty conception of time and place and a specially deficient memory for recent events. Moreover she frequently has what are euphemistically called "pseudo-memories," so that "the truth is not in her." This is one of the toxic varieties of insanity, and is commoner in women than in men. Male alcoholics seldom show Korsakow's psychosis, but tend rather to suffer from the more violent and dramatic "delirium tremens," with the tremors, acute distress, and hallucinations of rats, devils, &c., so familiar to the lay writer.

Passing from the patient's mental condition, we should carefully observe and describe any fits, tremors, or other spontaneous abnormal movements that may be present. Disorders of speech and articulation should next be studied, and the cranial nerves examined in due order.

It will be observed that, in our scheme of case-taking, sensory functions are investigated before motor. As a matter of experience, this order of examination is found to be of considerable practical advantage. The discovery of an area of anæsthesia often puts us rapidly on the track of a correct diagnosis and enables us to select with greater ease the salient points in the motor and other phenomena.

Certain accessory methods of examination, such as the testing of electrical reactions and lumbar puncture, are required only in special circumstances, where they may throw a flood of light on an otherwise obscure case. Inspection, palpation, and percussion of the skull are of considerable value in some cases of tumour of the brain, especially in cases of cerebellar growths. Sometimes it is advisable to have the scalp shaved, in order to detect abnormalities in the shape of the cranium.

Having collected our facts, comprising the history and the present state of the patient, we are now in a position to make our diagnosis. And in the process of diagnosis we have, first of all, to ask ourselves—Is the disease an organic one, due to a gross irritative or destructive lesion in the nervous system, for example cerebral hæmorrhage, alcoholic neuritis or tuberculous meningitis? Or is it one of the so-called "functional" diseases, that is to say, without known morbid anatomy, for example hysteria, migraine, neurasthenia?[1]

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[1] The boundary-line between functional and organic diseases is not so definite as might at first sight be supposed. At the present day, many

If the evidence points to an organic lesion, we have then to ask ourselves two further questions: (1) Where is the lesion? (2) What is its pathological nature? The answer to the first question, which constitutes the *anatomical diagnosis*, is derived mainly from a study of the distribution and grouping of the signs and symptoms. The answer to the second, constituting the *pathological diagnosis*, is attained mainly by a study of the history of the mode of onset.

In anatomical diagnosis we should always endeavour to think of a single lesion which will account for all the symptoms. Thus, for example, if a patient comes to us with hemiplegia of the left arm and leg, of the "upper motor neurone" type (see p. 22), and at the same time a right-sided facial palsy of the "lower motor neurone" type, instead of diagnosing two lesions, one in the right side of the brain causing left hemiplegia and another in the right facial nerve causing right facial palsy, we prefer to diagnose a single lesion in the right side of the pons, implicating simultaneously the right facial nerve and the pyramidal tract (see Fig. 5).

As an example of pathological diagnosis, suppose we have a patient with spastic paraplegia of both lower limbs and anæsthesia up to the level of the umbilicus, the anatomical position of the lesion is comparatively easy to fix, namely in the lower dorsal region of the cord, implicating both sensory and motor tracts. If the

diseases are classified as functional, for no better reason than that in them no constant microscopic changes have yet been recognised. In such diseases as epilepsy, paralysis agitans, and Raynaud's disease, there can be little doubt that profound molecular changes exist—in the two former instances in the central nervous system, in the latter in the sympathetic system—but these changes have not been recognised by the microscope. Other diseases again, due to poisoning by microbes or their toxins, or by other poisons—*e.g.* chorea, hydrophobia, tetanus, certain varieties of epileptiform fits, &c.—are undoubtedly the result of pathological changes affecting various groups of nerve elements. And yet, because these changes are not visible histologically, they are classed as "functional." Even hysteria itself, the prototype of functional diseases, has some profound underlying molecular change. The term "functional," then, is a confession of our etiological ignorance, and is by no means synonymous with "curable," as the steady and progressive advance of such a disease as paralysis agitans readily shows.

symptoms appeared suddenly, we think of a vascular lesion such as hæmorrhage or thrombosis; if they developed within a day or two, some inflammatory condition such as myelitis is probable; whilst if they only appeared very slowly, taking many months to reach their present intensity, we have to think of a slowly progressive lesion, such as a tumour.

We must never diagnose hysteria until we have excluded gross organic disease. And, finally, we should remember that the presence of certain hysterical symptoms does not exclude a co-existent organic lesion, or *vice versâ*. Hysteria and organic disease may be combined in the same patient, and this combination increases the difficulty of diagnosis.

LECTURE IV

COMA

We are not infrequently called to see a patient who is found to be unconscious. In such cases it is of great importance to make a correct diagnosis as to the probable cause. There are different degrees of unconsciousness. For example there are conditions in which the patient can be roused from his unconsciousness by shaking, shouting, or other stimuli, as in the case of ordinary sleep. When this degree of unconsciousness occurs in pathological conditions, as in a patient stupefied by various poisons (whether produced within the body or introduced from without), or from mechanical compression of the brain, for example by hæmorrhage, we call the condition *stupor*.

Coma is that degree of unconsciousness which is so deep that we are unable, by any ordinary stimulus, to rouse the patient. A deeply comatose patient does not swallow fluids placed in his mouth, his conjunctival reflexes are absent and his pupils insensitive to light.

How are we to proceed when called to see a patient whom we find comatose? Firstly, we inquire into the history, as to the patient's previous health, whether the coma was sudden or gradual in onset and whether it was preceded by other symptoms, such as convulsions or headache. We then examine the patient, feel the head for signs of injury, smell the breath, examine the pupils, noting their size, equality or inequality, and their reaction to light; we listen to the heart and note the character and frequency of the pulse and respirations. We observe whether the face is symmetrical or not, and whether there is conjugate deviation of the head and eyes in any direction. The optic discs in certain cases should be examined. We lift the limbs in turn and let them fall, observing whether there is any difference between the flaccidity of the two sides. We also test the knee-jerks and examine the abdominal and plantar reflexes on both sides. Then we pass a catheter, draw off the urine, note its specific gravity and test it for albumen and for sugar. Finally we note the temperature in both axillæ, and in certain cases we perform lumbar puncture and examine the cerebro-spinal fluid.

The first question is whether the coma is due to a general toxæmia, such as poisoning by alcohol or opium, uræmia, diabetes, &c., or whether it is the result of some gross intra-cranial lesion, such as hæmorrhage, meningitis, abscess, tumour, &c.

As a general maxim we may state that, if coma be toxæmic in origin, practically all the signs and symptoms will be bilaterally symmetrical. On the contrary, most cases of gross intra-cranial disease being unilateral, or at least asymmetrical, will therefore produce a corresponding preponderance of symptoms on one side of the body, so that, in addition to coma, we have a number of unilateral signs. Let us consider these unilateral cases first.

The commonest case is that of **spontaneous cerebral hæmorrhage.** Here the onset of unconsciousness is generally sudden; the patient's face is flushed or cyanosed, his skin sweats profusely, he breathes stertorously and his pulse is slow, full and bounding. All his limbs are flaccid, but on comparing the two sides, we find that the flaccidity is more absolute on the hemiplegic side. For example, the elbow of the affected side can be passively flexed to a greater degree than that of the healthy side. The arm and leg on the paralysed side also fall more "dead" than do those of the sound side when lifted up in turn and allowed to drop. The paralysed leg lies extended, whereas the healthy one tends to be semi-flexed. The head and eyes are often turned to one side, generally away from the paralysed limbs, unless the hæmorrhage be cortical or intra-ventricular, in which cases the deviation may be toward the paralysed limbs, and is associated with other irritative phenomena, *e.g.* spasticity instead of flaccidity. The face is asymmetrical, especially its lower part, the paralysed cheek flaps loosely during respiration and the mouth is distorted like a mark of exclamation laid on its side (!--), as if the patient were "puffing his pipe" at the paralysed angle of the mouth. The pupils are generally dilated and sometimes unequal, the larger pupil being on the side of the brain lesion. In pontine hæmorrhage, however, the pupils are often contracted to pin-points. At the onset of an ordinary apoplexy, whilst we get little help from the deep reflexes, which may or may not be diminished or lost on the affected side, there is, from the very outset, an extensor plantar reflex in the toes of the hemiplegic foot, and all the other superficial reflexes on that side are diminished or absent. The skin of the abdomen can be pinched or pricked on the paralysed side without eliciting an abdominal reflex—(Rosenbach's sign). The temperature on the paralysed side is usually higher than on the other, although the general temperature of the whole body falls at first. If the coma has lasted several hours, the bladder becomes distended and may develope an overflow incontinence. Afterwards, the temperature rises above normal, and in bad cases may go on to hyper-pyrexia.

Most cases of spontaneous cerebral hæmorrhage occur in patients past middle-age, in whom the arteries are no longer elastic and healthy, and there is often a history of kidney disease, with its resultant cardiac hypertrophy and high-tension pulse, conditions particularly liable to result in the bursting of a cerebral artery. The actual attack of hæmorrhage not uncommonly occurs during some slight physical exertion or mental excitement, as in public speakers, such as clergymen, politicians, or after-dinner orators, or in old men with brittle arteries, during straining at stool. In most cases of intra-dural cerebral hæmorrhage from whatever cause, the cerebro-spinal fluid is tinged with blood in greater or smaller amount (see later, p. 353).

But cerebral hæmorrhage may also, in rare cases, occur in young people having healthy vessels, as for example in a child during violent convulsions or during a paroxysm of whooping-cough, where the hæmorrhage is commonly venous and due to passive congestion with rupture of the cortical veins, or it may occur in any of the so-called "bleeding diseases"—purpura, hæmophilia, leukæmia, &c.

Cerebral hæmorrhage often occurs in cases of general paralysis of the insane, and in fact may be the first symptom calling attention to the disease. The symptoms are those already described, but there is usually a history of previous mental failure, grandiose ideas, loss of memory, attacks of emotional excitement, and slight indistinctness of articulation. In the absence of such history, we may be unable at the time to diagnose anything more than the fact of a cerebral hæmorrhage. But afterwards, when the patient recovers from his apoplexy—and the general paralytic recovers much more rapidly than the non-insane patient—we can generally recognise the characteristic evidences of the disease, both psychical and physical.

Coma may also be due to **traumatic cerebral "compression,"** where there is a hæmorrhage on the surface of the brain, either intra- or extra-dural. The signs are practically the same as in spontaneous apoplexy but the onset is different, for there is a history of a head injury. The symptoms develope gradually, especially if the hæmorrhage be extra-dural, beginning with local paralysis and perhaps localised convulsions. The paralysis gradually increases, the patient becomes drowsy, stupid, and finally comatose. There may be, before the onset of coma, a "lucid interval" of several hours or even a whole day, during which the patient, who was perhaps only stunned by the original blow, recovers consciousness and is apparently normal. A lucid interval, when followed by the above symptoms, generally indicates that the hæmorrhage is extra-dural. It is uncommon for symptoms of compression to come on immediately after the head injury, and when they do so, they suggest a depressed fracture pressing directly on the brain. And this can generally be detected by examination of the cranium.

Pontine Hæmorrhage is generally near the middle line, and therefore tends to produce bilateral symptoms. Pontine cases generally (but not always) have strongly contracted pupils owing to irritation of the third nerve nuclei. There is often hyperpyrexia and most cases are fatal.

Thrombosis of the Cerebral Sinuses is a rarer cause of coma. Here the diagnosis rests chiefly on the history. Cases secondary to suppurative conditions of the middle ear or frontal sinuses will have a corresponding history and the other signs of intra-cranial venous obstruction. Primary thrombosis of a sinus, occurring without infection, as in marasmus, profound anæmia, &c., is excessively difficult to recognise. Thrombosis of cerebral arteries, producing cerebral softening, often causes hemiplegia, but its onset is slower than is that of hæmorrhage; it more often comes on during ordinary sleep and is rarely associated with coma.

Amongst the other gross intra-cranial diseases producing coma, there is **cerebral meningitis.** Here again our diagnosis depends on the history. Most frequently the patient is a child. Instead of a history of sudden coma or of head injury we learn that there have been, for some days, headache, vomiting, photophobia and head-retraction, and often the characteristic "hydrocephalic cry." The child gradually becomes drowsy, apathetic, and finally comatose. Rigidity of the neck muscles and head-retraction persist during the coma (Fig. 21). Kernig's sign is often of value. This consists in a reflex contraction of the hamstring muscles when an attempt is made to put the sacral nerve-roots on the stretch by flexing the hip and at the same time extending the knee. If the meningitis be chiefly on the convexity of the brain, there are commonly convulsions preceding or accompanying the coma. On the other hand, if the meningitis be mainly basal, there are cranial nerve paralyses, especially of the ocular muscles. Examination of the cerebro-spinal fluid obtained by lumbar puncture gives conclusive evidence in cases of suspected meningitis. Optic neuritis, if

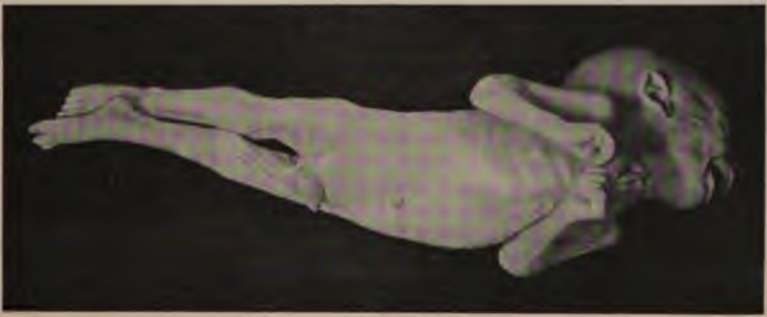

FIG. 21.—Case of posterior basic meningitis, showing head-retraction and posture of limbs.

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present, will indicate that the coma is not due to mere hæmorrhage, but that there is increased intra-cranial pressure, either due to meningitis, **cerebral abscess**, or possibly, if the history be a matter of weeks or months, to **intra-cranial growths**, syphilitic, tuberculous, or neoplastic.

Let us now pass to the other class of cases of coma, due not to a gross intra-cranial lesion, but to some general toxic condition of the higher cerebral centres. In this group the important point to notice is the absence of unilateral signs.

In the coma of **opium poisoning** there may be a history of laudanum swallowed or morphine injected hypodermically. An empty laudanum-bottle or a hypodermic-syringe may be found by the patient's side when he is discovered. If laudanum has been taken by the mouth, its odour may be detected in the breath. A chemical analysis of the stomach-contents obtained by the stomach-tube, will also help in the diagnosis. In the comatose patient we notice the excessive slowness of respiration, the slow and feeble pulse, the cold clammy skin, and, most striking of all, the pupils contracted to pin-points. These symptoms might be confused with those of pontine hæmorrhage. But there is not the pyrexia of a pontine apoplexy, and the coma of opium is not so deep as that of hæmorrhage. Moreover, in opium-poisoning the plantar reflexes are of the normal flexor type.

The coma of **acute alcoholic poisoning** is not so deep as that of apoplexy, for the patient can generally be roused, temporarily at least, by energetic stimulation. The typical stertor of apoplexy is not present, the pupils are dilated and react to light, and the corneal reflexes are preserved. The temperature is subnormal, the breath and stomach-contents smell of alcohol, and if we mix a specimen of the urine with potassium-bichromate solution and then allow strong sulphuric acid to flow to the bottom of the test-tube, a green colour appears if alcohol be present in the urine.

We must be careful, however, not to diagnose alcoholic poisoning simply because the patient's breath smells of alcohol. In the first place, a patient with cerebral hæmorrhage may have had alcohol given him, just at the onset of his symptoms. Or secondly, a patient who has been drinking alcohol may have an attack of apoplexy, or he may fall and sustain a head injury causing compression. Therefore every patient with apparent alcoholic coma should be carefully watched for about twenty-four hours, in case unilateral paralysis, or inequality of the pupils should supervene.

Post-epileptic Coma may be mistaken for apoplexy, if we do not happen to know that the patient is epileptic. But generally we have the history of preceding fits, and in a chronic epileptic there may be old scars about the scalp, tongue, or face, the result of injuries during previous fits. There is no preponderance of unilateral symptoms after a general epileptic fit, nor is there inequality of the pupils. The tongue may have been bitten during the fit and may be still bleeding during the stage of coma. Within an hour or less, the epileptic recovers consciousness without paralytic sequelæ.

Uræmic Coma is not uncommon, occurring as it does in patients who are the subjects of nephritis, acute or chronic. The coma is usually preceded by uræmic headaches, vomiting, and convulsions, local or general. A history of previous renal disease is here of great value. And there may be obvious signs of renal dropsy about the face and legs. The breath often has a urinous odour, and a catheter specimen of urine will show albumen together with various kinds of casts, and, in acute nephritis, blood. But we must not forget that a patient with chronic renal disease, a high-tension pulse and a hypertrophied heart, is just the one in whom cerebral hæmorrhage may fairly be expected. Therefore in a case of coma, mere albuminuria should not lead us to diagnose uræmic coma. We must always be on the look-out for symptoms of unilateral paralysis.

Diabetic Coma is easy to recognise, if we know that the patient has been suffering from diabetes. Even if there be no history of diabetes, examination of the urine shows the characteristic high specific gravity, together with the presence of sugar in large amount as shown by the ordinary tests. And the addition of a few drops of liq. ferri perchloridi to the urine produces a deep brownish-red colour, due to di-acetic acid. The "acetone" smell of the breath is unmistakable and only occurs in diabetic patients.

Further, diabetic coma is not sudden in onset, but is commonly preceded by headache, irritability and uncontrollable drowsiness, merging into profound coma with remarkably deep noisy breathing. The pulse is usually small and rapid, unlike the full, slow pulse of cerebral hæmorrhage.

Sunstroke sometimes causes coma. Here, of course, it is essential that there should be a history of exposure to a hot sun, of a previously healthy patient. Alcoholic patients are more liable to sunstroke than teetotalers. A patient who is comatose from sunstroke often has extraordinary hyperpyrexia—108° F. and upwards. General convulsions may occur.

In malarial climates we must also be prepared to meet with a comatose variety of **pernicious malaria**, where the parasites produce thrombosis of the smaller cortical vessels. A malarious patient may rapidly become comatose and die within a few hours, as happened to a friend of my own. In such cases the malarious history and the examination of the blood for the plasmodium will settle the diagnosis. A patient dying from cancer may become comatose shortly before death—the so-called **coma carcinomatosum.**

We need not do more than mention the terminal coma of such diseases as acute yellow atrophy, or the "coma-vigil" of typhus and of severe enteric fever.

Hysterical Trance, by a careless observer, might be mistaken for true coma. The hysterical patient, however, has neither stertor nor cyanosis, the breathing and heart's action are regular, though perhaps very faint, the pupils react to light and the patient generally resists forcible opening of the eyes. Thus a young man of twenty-two who had sudden attacks of apparent sleep (narcolepsy) coming on in the middle of meals or when playing cards, refused to be roused by ordinary stimuli in the form of shaking or shouting, but yielded at last to forcible digging in the ribs. He passed through a stage of "grande hysterie" before waking up, and for some time afterwards had hysterical blindness and other hysterical stigmata.

The **hypnotic trance** may be regarded as an artificially-induced form of hysteria.

LECTURE V

FITS AND OTHER CONVULSIVE PHENOMENA

We are often consulted about patients who are said to have "fits," but we are seldom fortunate enough to witness an attack. If we do, the diagnosis presents little difficulty. More often, in making the diagnosis as to the nature of a fit, we have to depend upon the description given by the patient's friends.

The following is a list of the chief clinical conditions in which convulsive phenomena occur.

- CEREBRAL FITS
  - HYSTERICAL
    - Hysteria major.
    - Catalepsy.
    - Hysteria minor.
    - Post-epileptic hysterical attacks.
  - EPILEPTIFORM
    - Epilepsy major (*grand mal*).
    - Epilepsy minor (*petit mal*).
    - Post-epileptic automatism—"masked epilepsy."
    - Toxic conditions:—Uræmia, puerperal eclampsia, alcohol, absinthe, lead, &c.
    - General paralysis of the insane.
    - Organic brain lesions—Jacksonian fits, &c.
    - Infantile Convulsions
      - Toxic.
      - Organic.
    - Stokes-Adams' disease.
- CEREBELLAR FITS.

In making inquiries about convulsive phenomena, whatever be their nature, it is advisable to avoid using the word "fit," especially if we are discussing symptoms in the presence of the patient. It is better to refer simply to "attacks." Many epileptics are unaware of their own disease, and even when they know its nature, they dislike hearing about their "fits."

The first point we should try to determine is whether the attacks are hysterical or epileptiform. In reference to this the age and sex of the patient are often of importance. We do not meet with hysteria in infants, and rarely in children below the age of puberty. Epilepsy is of equal frequency in both sexes, whereas hysteria is twenty times commoner in females than in males. Hysterical attacks in male patients are most frequent in lads at about the age of puberty.

Scheme of Investigation in Cases of Fits

Exciting cause. Nature of warning, if any. Onset, sudden or gradual. Scream. Injuries during falling. Movements, tonic, clonic, purposive. Starting-point of movements, and exact order of spread. Biting of tongue. Micturition or defæcation. Colour of face, pale, flushed, cyanosed. Pupils. Conjunctival reflexes. Knee-jerks immediately after attack. Duration of attack. After-symptoms (coma, vomiting, headache, sleep, &c.).

Pursuing the foregoing scheme, we should inquire whether there was any apparent cause for the attack. Hysterical attacks generally follow some emotional disturbance. **Epilepsy** comes on without exciting cause. A warning or *aura* before an attack of epilepsy may be of the most varied character. Perhaps the commonest is the "epigastric" aura, or there may be an indescribable feeling of terror, subjective auditory or visual phenomena, visual auræ being most frequently red in colour, unilateral tingling or twitching of the face or one of the limbs, or a "dreamy" mental state (sometimes associated with subjective sensations of smell or taste), and so on, according to the particular cortical area whence the epileptic explosion happens to start. But often the epileptic has no aura, he falls suddenly as if struck down by an unseen hand. On the contrary, **hysterical attacks** usually come on gradually, and are often preceded by the hysterical "globus" or ball in the throat, or by feelings of palpitation, excitement, giddiness, tingling in the feet, &c. Sometimes at the onset of the attack the epileptic, as he falls, utters a weird epileptic cry or moan, which is not repeated. By this time he is already unconscious and does not hear his own cry. Frequently he injures himself in falling, especially by striking his head. Many chronic epileptics may be recognised by the presence of numerous scars on the scalp and face. The hysterical patient, on the contrary, never injures herself when falling. She comes down carefully, often on a sofa or easy-chair. She not uncommonly screams, and may continue to scream or shout throughout the attack.

The nature of the movements during the attack is of importance. In epilepsy we have the tonic stage, in which all the voluntary muscles, including those of respiration, become suddenly rigid. The patient, therefore, falls like a log, and his lips and face become cyanosed, his pupils dilating and not reacting to light. The tonic stage passes into the clonic, in which violent jerking occurs in all the voluntary muscles, at first rapid, and gradually becoming slower and of greater range. The eyes, which during the tonic stage had been drawn to one side (the side on which the tonic spasm was more intense), now show rapid clonic jerks towards that side. The face loses its cyanotic hue, air re-enters the lungs, and is jerked out in short puffs mixed with saliva, forming a froth which is not unfrequently blood-stained, since the jerking tongue may be bitten by the clonic movements of the jaws. During this stage the patient often passes urine and fæces. Within two or three minutes from their onset, the movements gradually cease, and the patient remains in a state of coma, with stertorous breathing and flaccid limbs, the eyes being now turned to the side opposite to that towards which they originally deviated. Then, after ten minutes or so, the coma passes off, and the patient may either wake up, perhaps with a headache, or he may vomit, or he may pass into a sound sleep.

In a hysterical attack, on the other hand, the patient's face is natural in colour, never cyanosed, though later it may become flushed from physical exertion. The voluntary muscles are usually contracted, the fists clenched, the eyes tightly closed and resistant to opening, but if the eyes be forcibly opened the eyeballs roll upwards. Then, after a stage of general tremor totally unlike the clonic stage of epilepsy, the patient makes violent "purposive" movements, kicking, pushing, biting, rolling about, banging her head on the floor, beating her own face, pulling her hair, &c. During this stage various grotesque postures may be assumed; of these, the most characteristic is one in which the back is arched (opisthotonos) and the patient rests on her head and heels. Or there may be curving of the trunk laterally (pleurosthotonos), or forwards (emprosthotonos), "crucifixion" attitude, &c. The patient may talk, scream, or sing during the attack, which may last for many minutes. But throughout the attack the pupils generally react to light, and the conjunctival reflex is usually preserved. The hysterical patient never bites her tongue, though she may bite her lips or fingers, or snap at the fingers of bystanders. She never empties the bladder or rectum during the attack, and after it is over she may or may not have any recollection of what has happened, and is sometimes in a semi-dazed condition.

The knee-jerks, during the stage of flaccid coma terminating a severe epileptic fit, may be temporarily abolished, but soon they become exaggerated, and, for a few minutes, ankle-clonus may often be elicited. In hysteria the deep reflexes are unaltered.

If we bear in mind the foregoing points, the diagnosis between a severe epileptic fit and an attack of "grande hysterie" is generally easy.

Catalepsy, another variety of hysterical attack, is easily recognised by the peculiar immobility of the limbs. The patient during the attack, though not unconscious, is unable to move a muscle, but her limbs are plastic like those of a lay figure, and if placed passively in any posture, remain there.

There are also many varieties of *minor hysterical attacks*, easy of recognition, of which the commonest consist merely in emotional outbursts of uncontrollable laughing or crying, or a feeling of a lump in the throat—"globus hystericus"—which causes the patient to swallow.

Supposing, then, that we have come to the conclusion that the patient's attacks are hysterical and not epileptiform, we should not rest content until we have settled the further point as to whether the hysterical attack was preceded by an attack of minor epilepsy or *petit mal*.

Attacks of *petit mal* are often overlooked. It is well to remember that in true epilepsy (whether major or minor) the one essential phenomenon is not convulsions but loss of consciousness. In *petit mal* it may be the only phenomenon, so transient, perhaps, that the patient does not even fall; he simply pauses for an instant during conversation, looks strange, and then goes on with what he was saying. Or he may fall down and get up again immediately, a variety of epilepsy which is often mistaken for syncope, but is distinguished by the suddenness of onset and of recovery. If we happen to observe a patient at the moment of his attack of *petit mal*, we generally notice that the pupils dilate and his face turns momentarily pale, the pallor being followed by flushing. It is immediately after such a minor attack that some patients go on to a *post-epileptic hysterical attack*, and if the initial epilepsy be not recognised, treatment will fail.

Attacks of minor epilepsy are, now and then, associated with *post-epileptic automatism*, in which the patient has an attack of *petit mal* which perhaps passes unnoticed; he then proceeds to perform some unusual or inappropriate act, of which he has afterwards no recollection. Perhaps the commonest automatic action is that of undressing; or he may proceed to empty his bladder, as in the oft-quoted instance of the judge who did so in the corner of his court; or he may perform some still more complicated action. Thus a case of mine was that of a well-known financier who had several attacks of loss of memory. During one of these, lasting two and a half hours, he attended an important board-meeting and proposed certain resolutions to which, both before and after, he was strongly opposed. He then took a friend out to lunch and finally returned to his office. He then woke up and asked his confidential clerk where he had been. The medico-legal significance of these cases is of importance, since such a patient, in a condition of post-epileptic automatism, may commit serious and complicated crimes, of which he has afterwards no recollection. A previous history of epilepsy, either major or minor, is here of great diagnostic value.

Supposing that, having excluded hysteria, we arrive at the conclusion that a patient's fits are epileptiform, we must still remember that other conditions besides idiopathic epilepsy can produce epileptiform fits. Thus for example fits may be of toxic origin. Acute *alcoholic* or *absinthe poisoning* may produce coma and convulsions. The history and the smell of the breath will usually guide us in such cases. The convulsions of *lead poisoning* are usually accompanied by other signs of plumbism, such as the blue line in the gums, weakness of the extensors of the wrist, optic neuritis, &c. Toxins produced within the body may also cause fits exactly similar to epilepsy, witness the *uræmic convulsions* of Bright's disease and of puerperal eclampsia. In every case of fits appearing in a previously healthy individual, the urine should be tested and the optic discs examined. In the case of a young officer who was brought into hospital at Pretoria suffering from a succession of fits typically epileptic in character, but simply uræmic, acute nephritis was the cause. In chronic renal disease, besides the cardio-vascular changes, there not uncommonly exists albuminuric retinitis, which is of great diagnostic significance. When we come to the subject of infantile convulsions we shall find that many of them are toxic in origin.

Epileptiform fits may occur during the course of *general paralysis of the insane;* they may, in fact, be the first symptom of the disease. Epileptic fits appearing for the first time in a middle-aged patient should always suggest the possibility of paralytic dementia. In such cases we look for inequality or irregularity of the pupils, and especially loss of the light reflex, mental changes, facial tremors, and slurring articulation, and a history of syphilis should be sought. Lumbar puncture may show lymphocytosis of the cerebro-spinal fluid, a condition which is constant in general paralysis but does not occur in idiopathic epilepsy.

Intra-cranial tumours anywhere, even deep within the substance of the brain, may cause general epileptiform fits, from *increased intra-cranial pressure.* Here we are usually guided by the cardinal signs of intra-cranial tumour—headache, vomiting, optic neuritis, &c.

All the epileptiform fits to which we have as yet alluded have a bilateral general distribution, and are not succeeded by any localised paralysis. But when fits are produced by gross focal irritation of the cortex their onset is a local one, and they are not necessarily associated with loss of consciousness. Such "*Jacksonian*" *fits* are usually followed by weakness of the part which is primarily convulsed. Jacksonian fits may occur as often as fifty or a hundred times a day. They usually begin with a subjective sensory aura, such as tingling, numbness or twitching, localised in some particular part, *e.g.* the thumb or big toe. Then there is tonic spasm of that part, followed by clonic jerking. This may remain confined to the muscles where it began, or it may spread to others. If it spreads, it does so by a deliberate march from one cortical centre to another (Fig. 3, p. 4). Thus a fit commencing in the big toe would occur successively in the ankle, knee, hip, shoulder, elbow, hand, &c., and would affect the face last of all. Or a fit beginning in the elbow would spread *viâ* the shoulder, hip and knee, to the toes, and simultaneously *viâ* the wrist, fingers and neck, to the face and tongue, as the ripples produced by dropping a stone into a pool spread concentrically in ever-widening circles. A patient who has Jacksonian fits may remain conscious throughout the fit and may even be able to speak, though he is usually somewhat excited. But if the convulsion spreads to the opposite cortical area, thus

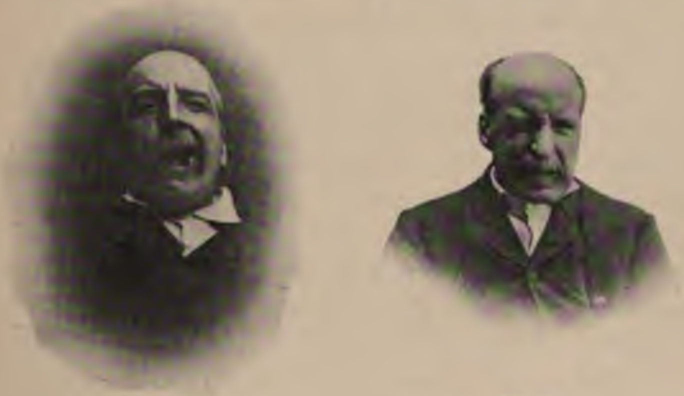

FIG. 22. FIG. 23.

Cortical gumma of the face-centre of the right cerebral cortex.

Fig. 22 shows patient during a Jacksonian fit of the left face.
Fig. 23 shows weakness of the left face on voluntary movement.

becoming bilateral, consciousness is lost as the fit crosses over. Jacksonian fits are followed by local weakness and increased deep reflexes in the convulsed part. Fig. 22 shows a patient during a localised fit in the left face, due to a gumma in the cortical facial centre. Fig. 23 shows the maximum voluntary movement of the face after an attack. It will be seen that the left lower face is markedly weaker than the right. The localised paralysis passes off in the reverse order from that in which the spasm appeared, the muscles first convulsed being the last to recover power.

Of course, cortical lesions will produce localised motor phenomena only if they affect the motor centres in the region of the Rolandic fissure. A focal lesion of a sensory cortical area produces, not a motor, but a *sensory fit*. Thus disease of the tip of the temporo-sphenoidal lobe (Fig. 4, p. 4) causes a sudden subjective sensation of smell or taste (often associated with a characteristic "dreamy" mental state), occipital lesions cause subjective visual hallucinations such as flashes of light, and so on. Moreover, after the sensory fit it is not uncommon to find temporary sensory paralysis, *e.g.* anosmia after a temporo-sphenoidal, or hemianopia after an occipital fit.

A Jacksonian fit, of whatever variety, is the result of a local lesion in the neighbourhood either of the cortex or of its superjacent meninges or bones. If the lesion be in the substance of the cortex, not merely superficial to it, there will be some local paralysis even before the fit occurs.

Localised fits may be produced by any irritative cortical lesion. The commonest causes are tumours, syphilitic or otherwise, abscesses, meningitis of any variety, local hæmorrhages, depressed fractures, and so on. Localised fits may also be produced by sub-cortical tumours in the pre-central region. In such cases we observe muscular paresis in the affected limb, together with recurring convulsive phenomena in the limb; but the point of onset of the successive fits is less constant than in a true cortical convulsion, and the fit begins sometimes in one, sometimes in another muscle-group of the affected limb.[1] We should also bear in mind that Jacksonian fits may occur in certain cases of general paralysis of the insane, and sometimes even in uræmia.

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[1] *Cf.* van Valkenburg, *Neurologisches Centralblatt*, 1906, p. 594.

Infantile Convulsions are epileptiform attacks occurring in infancy. The symptoms are similar to those of true epilepsy, but less violent. When called to see a child with convulsions, we should first examine for rickets, since rickety and hereditarily neurotic children are especially prone to convulsions. And we should also try to determine whether the fits are reflex, toxic, or organic in origin. In rickety infants of neurotic inheritance *reflex convulsions* may be set up by peripheral irritation such as the cutting of a tooth, round-worms in the intestine (thread-worms do not cause convulsions), a tight prepuce, &c. Moreover, rickety children often have other nervous symptoms, such as tetany, laryngismus stridulus, and the well-known inversion of the thumbs towards the palms, a phenomenon which sometimes precedes a convulsion. *Toxic* conditions may produce convulsions in previously healthy children. Thus an acute fever such as pneumonia, measles, scarlet fever or influenza, may be ushered in by a convulsion instead of by a rigor as in the adult. The convulsions produced by round-worms may be partly toxic in origin. Uræmic convulsions occur in children, though less often than in adults, and in cases with recurring convulsions the urine should always be examined. Asphyxial convulsions, due to deficient oxygenation of the brain, occur in dyspnœa from any cause, *e.g.* during pneumonia and diphtheria, during a paroxysm of whooping-cough, or in the cyanosis of congenital heart disease. Intra-cranial *organic* lesions may cause infantile convulsions. Thus, for example, polio-encephalitis superior, an acute inflammatory affection of the cortex, has a febrile onset with vomiting and convulsions, usually more marked on one side than on the other. After the convulsions have passed off, the child is often left permanently hemiplegic or diplegic. If the cortex of the frontal region is affected, permanent mental dulness may remain. Local disease or injury of the bones or membranes may produce infantile convulsions. Convulsions appearing within a few hours after birth are not unfrequently the result of compression of the brain by a meningeal hæmorrhage.

Meningitis, whether due to the tubercle bacillus, to syphilis, or to other organisms, may produce convulsions, not only when cortical, in which case convulsions appear early, but also in basal cases, where the fits are due to increased intra-cranial pressure.

Epileptiform fits also occur in one variety of the **Stokes-Adams' syndrome**, a condition which occurs after middle life, usually in male patients with degenerate arteries, and which is characterised by paroxysmal attacks of abnormal slowness of the heart beat, the rate sinking as low as twenty per minute or even less. Such a patient is liable to syncopal attacks, to which may be superadded epileptiform fits or attacks of coma without convulsions. More or less slowness of pulse usually persists between the paroxysms, as a permanent phenomenon.

Strychnine Convulsions should never be confounded with epileptiform fits, since they begin with clonic spasms, and shortly become tonic with opisthotonos, the tonic spasms recurring again and again with increasing severity. A patient poisoned by strychnine, however, is not unconscious. Moreover, there are periods of intermission, lasting for several minutes at a time, during which the muscles are relaxed. The tonic spasms of **tetanus** might be mistaken for strychnine-poisoning, but they have no initial clonic stage. The earliest symptom of tetanus is the well-known tonic spasm of the jaw muscles, producing "lock-jaw." To this there are superadded paroxysms of tonic spasm in the face (*risus sardonicus*), trunk and limbs, with opisthotonos, somewhat like those of the tonic stage of strychnine-poisoning. Between the tetanic paroxysms there is no complete muscular relaxation as in strychnine-poisoning, but simply a remission in the degree of spasm, the jaw muscles remaining partially contracted.

A patient suffering from **rabies** exhibits great mental excitement, with tonic spasm of the muscles of deglutition, especially on attempting to swallow liquids (hence the misnomer hydrophobia). The spasm may also be induced by other stimuli such as a bright light or a loud sound. The spasm spreads to other muscles, especially those of respiration, and severe opisthotonos may supervene at the end, the patient dying either of respiratory spasm or from syncope. Hydrophobia may be simulated by hysterical patients who have been bitten by a non-rabid dog, and in such cases globus hystericus and hysterical opisthotonos may both occur, but true respiratory spasm does not occur in hysterical attacks, though there may be hysterical rapidity of breathing.

During an attack of **tetany** the posture is very characteristic. There is a bilateral tonic spasm, usually painful, of the hands and feet, the hand assuming a conical shape, the fingers being extended at the inter-phalangeal joints, slightly flexed at the metacarpo-phalangeal joints and pressed together with the thumb tucked inside the fingers; at the same time the muscles of the thenar and hypothenar eminences are contracted, so that the hollow of the palm is deepened. In the feet, the toes are flexed towards the sole, the ankle is dorsiflexed and the foot is sometimes inverted. These postures may persist during sleep. Pressure on the nerve-trunks

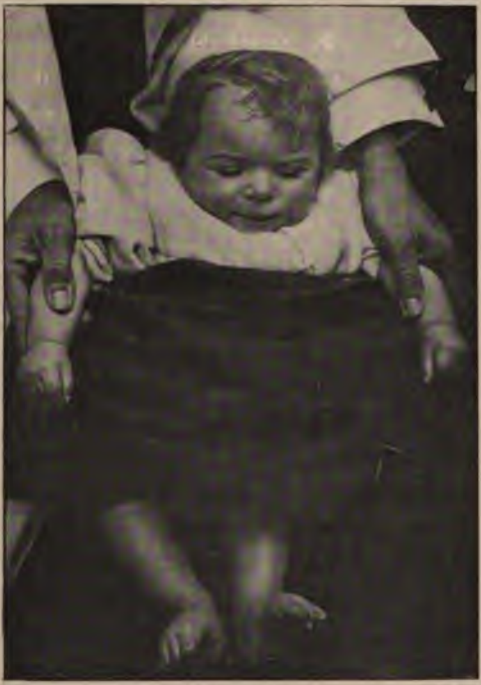

FIG. 21.—Tetany.

of the affected limb induces a typical spasm (Trousseau's sign), and the muscles and nerves are unduly irritable both to faradism and to galvanism (Erb's sign). Tetany is most commonly met with in rickety children (Fig. 24), in whom it is often associated with laryngismus stridulus and with excessive irritability on percussion of the facial nerve (Chvostek's sign). But it occasionally occurs in adults, *e.g.* after extirpation of the thyroid gland, or rather of the parathyroids; sometimes during pregnancy or lactation; and also as a grave terminal symptom in dilatation of the stomach. Cataract is a curiously frequent concomitant of the gastric form of tetany. An endemic form of tetany also occurs in certain Continental towns, chiefly in the winter months. It is especially common amongst shoemakers.

Cerebellar Fits.—Irritative lesions of the cerebellum are occasionally associated with cerebellar fits. These are never clonic, but consist of tonic spasms, sudden in onset. In *unilateral* cerebellar disease the spasms are more marked in the homolateral limbs than in those of the opposite side. The face is usually unaffected. The homolateral limbs become rigidly adducted to the trunk, the contra-lateral limbs are abducted, whilst at the same time there is a screw-like rotation of the limbs, trunk, and head around their own long axes, from the side of the lesion towards the healthy side,[1] and a deviation of the eyes towards the healthy side. Hughlings Jackson has described another variety of cerebellar fit occurring in cases of tumour of the *middle lobe.* Here also, as in tumours of the lateral lobe, the fits are tonic, not clonic. They consist of head-retraction with arching of the back, flexion of the elbows, supination of the hands, and rigid extension of the legs, with pointing of the toes.

But let us not forget that epileptiform fits, cerebral in type, may also occur in cerebellar tumours, due either to the general increase of intra-cranial pressure, or to a fulminating meningitis superadded to an old tuberculous tumour.

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[1] Grainger Stewart and Holmes, *Brain*, 1904.

LECTURE VI

INVOLUNTARY MOVEMENTS

Besides fits, which we have already considered, there are many other conditions in which involuntary contractions occur in the voluntary muscles. But our knowledge of the mechanism of their causation is so incomplete that it is impossible at present to classify them accurately. We must therefore content ourselves, in the meanwhile, with referring to some of their chief clinical varieties.

In studying involuntary movements occurring in striated muscles, it is important to observe whether the movement is confined to an individual muscle or part of a muscle, or whether, on the other hand, it consists in alternate contraction of muscles and of their antagonists. We should also observe whether the abnormal muscular contractions produce movements of a joint, or whether they are so localised, either to a small muscle or to part of a larger one, that we merely see or feel the muscle fibres contracting beneath the skin.

The muscular phenomenon known as **shivering** or **rigor** is sometimes physiological. For example, when a healthy person becomes chilled, as by prolonged swimming, he often shivers on coming out of the water. The involuntary muscular contractions of which shivering consists are for the purpose of producing heat and thereby raising the depressed body-temperature. But often rigors are toxic in origin, as are those occurring at the onset of certain acute fevers. Thus we have rigors in pneumonia, ague influenza, scarlet fever, &c. And the rigor which sometimes follows catheterisation is probably also toxic in origin, since it rarely occurs except when there is a raw surface in a urinary tract which is not aseptic. In all these toxic rigors, although the shivering patient has a sensation of cold, his temperature meanwhile is rising, and it continues to rise until the rigor stops. He has the sensation of cold because by vasomotor action the blood is driven out of his skin, which is therefore cooled and is actually cold, although the temperature of the blood is raised.

Transient flickering or quivering of a muscle, a condition known as **myokymia** (or more popularly as "live flesh"), affecting a few muscle-bundles of a single muscle, without producing movement of a joint, is not uncommon in people who are anæmic or out of health. It is specially common in the orbicularis oculi and in some of the larger muscles of the limbs, *e.g.* the deltoid and biceps in the upper limb, the glutei and quadriceps in the lower. This variety of myokymia is not associated with muscular atrophy, nor with any alteration of electrical excitability. It is unaffected by rest or by voluntary exertion, and has no serious significance.

Sometimes, however, fibrillary movement occurs in organic lesions of the lower motor neurone. Thus in the muscular atrophy of *chronic anterior poliomyelitis*, of *amyotrophic lateral sclerosis*, and of some cases of *syringomyelia*, diseases in which the cells of the anterior cornua are undergoing slow degeneration, there may be seen fibrillary or fascicular tremors in the wasting muscles. This quivering myokymia can often be elicited by gentle flicking, or by a breath of cold air blown over the skin. A precisely similar fibrillary tremor occurs in the wasting tongue of *bulbar paralysis*, when the degenerative process has involved the hypoglossal nuclei. Fibrillary tremor does not occur in the primary myopathies, whether atrophic or pseudo-hypertrophic in type. In other cases fibrillary tremor occurs in the distribution of a motor nerve which has begun to recover from previous paralysis. It is not uncommon in the face during convalescence from facial palsy, and sometimes it persists for months and years after voluntary power has returned, as in the case of a professional friend of my own who is otherwise perfectly well. But usually the myokymia passes off when motor power has been restored.

Somewhat different in appearance is the condition known as **myoclonus**, a rare disease, characterised by paroxysms of sudden shock-like contractions in various muscles, lasting for several minutes at a time, irregular in rhythm and varying in rapidity from ten to fifty per minute. The muscles affected are usually those of the limbs, often symmetrically on the two sides, but contractions may also occur in the trunk and even in the face. Sometimes the diaphragm and the larynx are affected, so that curious grunting respiratory sounds are produced. There is no muscular atrophy or alteration in electrical excitability. The spasms cease during sleep. Two varieties of myoclonus have been observed; in one—Friedreich's *paramyoclonus multiplex*, usually a disease of adult life—the myoclonus ceases on voluntary movement. The other variety is Unverricht's *family myoclonus* or *myoclonus epilepticus*, in which several members of a family are affected, all belonging to the same generation, though the disease is not handed down from parent to child. In addition to the myoclonus, these patients have epileptiform fits, and they tend ultimately to become more or less demented. Moreover, the muscular contractions in family myoclonus are intensified by voluntary movement and by psychical excitement. Family myoclonus usually manifests itself in early life, often at or before puberty. Thus in a little boy of thirteen suffering from myoclonus affecting the muscles of the neck, shoulders, trunk, thighs and cremasters (see Fig. 25), the condition had existed since the age of four, and he had also epileptiform fits. Two elder brothers were similarly affected, both of whom, as the patient graphically put it, began to "click" at the age of seven. On the other hand, another patient suffering from Friedreich's paramyoclonus, showed no symptoms of the disease till the age of forty-seven. He never had fits, and at the age of fifty, when he came under observation, he had myoclonic shocks limited to the muscles indicated in the accompanying diagram (Fig. 26).

We next pass to the consideration of **tremors**. The term "tremor" is applied to involuntary rhythmic oscillations of one or more parts of the body, resulting from the alternate contraction of muscle-groups and of their antagonists. A *simple* tremor is one which affects a single muscle-group and its antagonists, whilst in a *compound* tremor several groups with their antagonists are in action, producing a complex movement, *e.g.* flexion and extension of the fingers combined with pronation and supination of the forearm. Tremors may be rapid or slow; they may be diminished or increased by voluntary exertion, and they generally cease during sleep.

We must not forget that an ordinary voluntary muscular movement is not the result of a continuous muscular contraction but is constituted by the fusion of a rapid succession of short single contractions, averaging from ten to twelve per second. In conditions of temporary *fatigue* or of chronic *asthenia* the rate of these muscle discharges becomes slower and less regular, so that the individual muscular contractions fuse less perfectly and the result is a very fine tremor. In *emotional* excitement—more often in

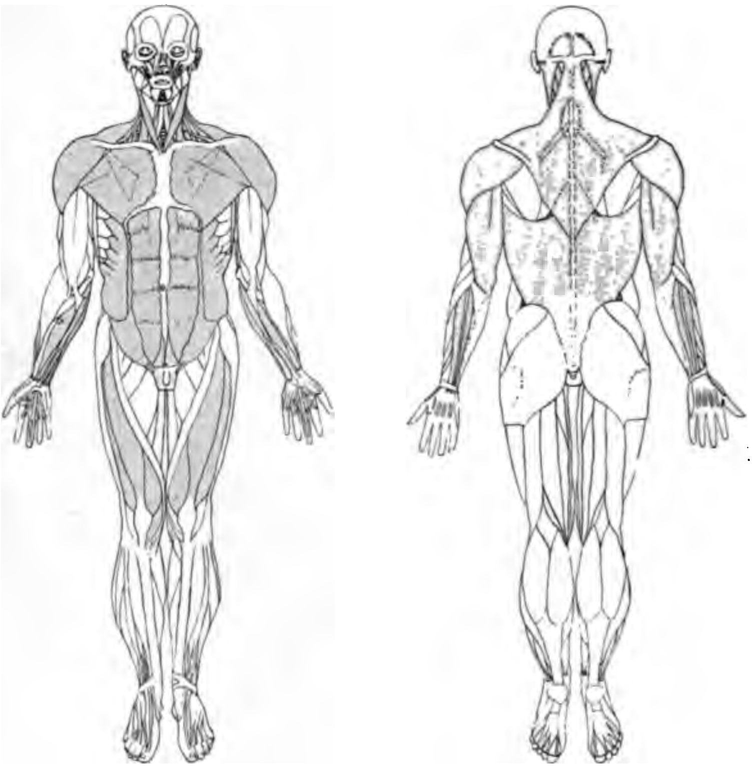

FIG. 25.—Unverricht's family myoclonus or "myoclonus epilepticus" in a boy aged 13. Besides the muscles indicated in the diagram, the soft palate was also affected.

states of fear than from pleasurable emotion—there may be a fine rhythmic tremor of about eight or nine oscillations per second, and the same occurs in *exophthalmic goitre*. This tremor is made more apparent by voluntary exertion. It is best elicited by making the patient hold his hands horizontally in front of him, with the fingers widely spread. We can then feel the tremor even better than see it, by placing our own fingers lightly on the dorsum of the patient's hand. This fine tremor affects all the limbs, and is familiar to the knees of many a public speaker.

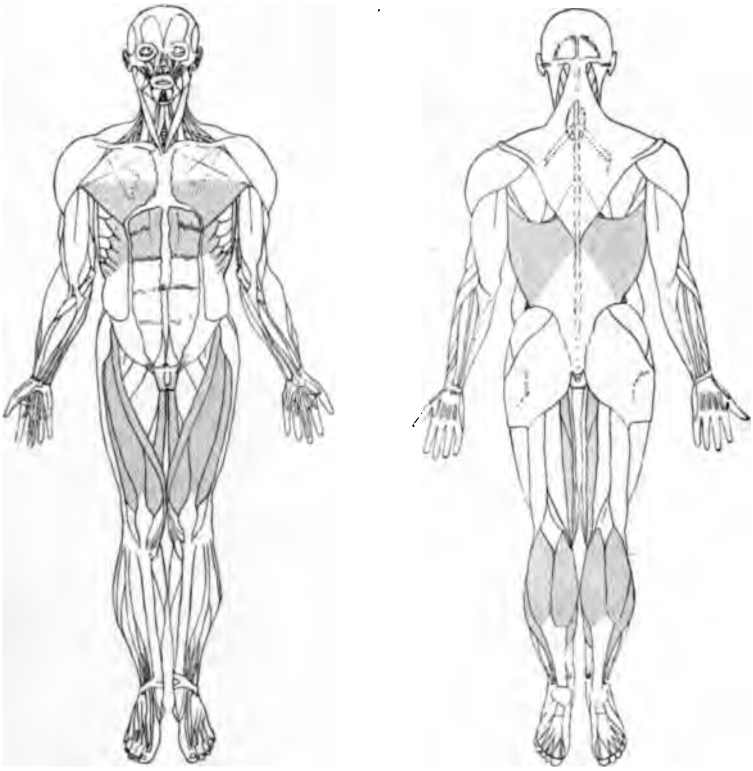

FIG. 26.—Friedreich's para-myoclonus multiplex in a man aged 50.

Similar fine tremors also occur in certain toxic conditions. The tremor of chronic alcoholic poisoning is familiar to the layman, and when an alcoholic patient becomes delirious and maniacal, his tremors are so evident that the condition is termed "delirium tremens." But other poisons besides alcohol produce fine tremors: for example, nicotine poisoning from excessive cigarette-smoking, also chronic poisoning by lead, mercury, chloral, &c.

In cases of suspected alcoholism a valuable corroborative sign, known as *Quinquaud's sign*, may often be elicited. In testing for this, we make the patient spread out his fingers and press them at right angles to the palm of our own hand, which we hold in a vertical position. For the first two or three seconds nothing particular is noticed, but if the patient be a chronic alcoholic, we soon begin to feel a series of slight shocks, as if the phalanges of each finger were knocking, one against the other, trying to reach our palm. If the phenomenon be present, the patient is almost certainly a chronic alcoholic.

The tremor of **paralysis agitans** is generally rather coarse, varying in different cases from four to seven oscillations per second. It produces joint movements, *e.g.* the well-known "pill-rolling" movement of the thumb and fingers, flexion-extension movement of the wrists, pronation-supination of the forearm, flexion-extension of the ankle, &c. The coarser the movement, the slower is the rhythm. Paralysis agitans generally begins unilaterally, and may remain confined to one side for some time before ultimately becoming bilateral, as may be seen in Figs. 27 and 28, which are taken from the same patient at an interval of three years. Usually the tremor of paralysis agitans can be temporarily controlled by voluntary exertion. But this is not always so, indeed cases occur in which voluntary movement increases the tremor. Paralysis agitans is always accompanied by rigidity in the affected muscles; in fact, rigidity may be well marked without tremor, in the so-called paralysis agitans "sine agitatione."

Senile tremor is not unlike the tremor of paralysis agitans, but its onset occurs much later in life. Moreover, it begins bilaterally, and is unaccompanied by the characteristic rigidity of Parkinson's disease. Thus in a famous old admiral it began at the age of eighty-four as a gentle antero-posterior tremor of the tongue, with a synchronous movement of the orbicularis oris.

Both his hands had tremor and an interosseal attitude like that of paralysis agitans, but without rigidity.

In rickety children, especially during the second six months after birth, we sometimes observe a peculiar involuntary rotatory or nodding tremor of the head which usually comes on quite suddenly in winter-time and is known as **spasmus nutans.** It is generally accompanied by nystagmus, which may be more marked in one eye than in the other. If we fix the child's head, the nystagmus increases. The head movement is more often a lateral rotation than an antero-posterior nodding. It stops when the child's eyes are closed, either voluntarily or artificially. The symptom persists for six or eight weeks or longer, and then passes off, perhaps to return again in the following winter. It is not associated with any mental defect.

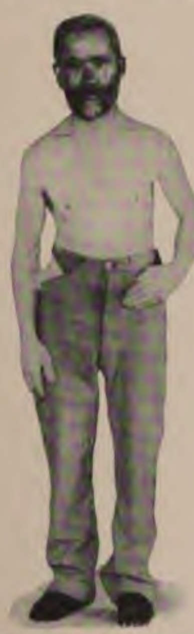

FIG. 27.—Paralysis Agitans, left-sided.

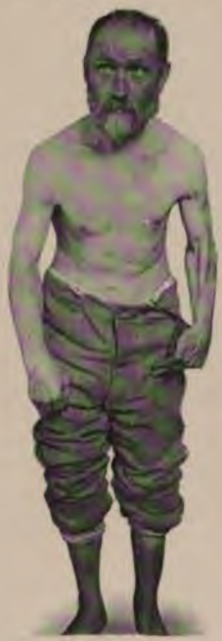

FIG. 28.—Paralysis Agitans:—the same patient as in Fig. 27, three years later.

Rhythmic tremor sometimes occurs in gross brain disease. Thus, for example, in cases of **lesions in the tegmental region** of the pons or crus cerebri,[1] affecting the *rubro-spinal tract* (which descends from the red nucleus into the lateral column of the opposite side), or in lesions of the adjacent part of the *optic thalamus*, we occasionally observe a slow rhythmic tremor of the affected limbs, chiefly the hand and foot. This tremor is increased by excitement or voluntary movement, but ceases during sleep. In the diagnosis of such cases we have, besides the tremor, the other localising signs of a gross destructive lesion. Thus if the ocular nuclei (which lie dorsal to the red nucleus) be affected, there is ocular palsy of the nuclear type; if the pyramidal tract be affected there is spastic hemiplegia; and if the sensory tract, traversing the internal capsule and optic thalamus, be implicated, we have hemianæsthesia. When a unilateral lesion of one crus cerebri produces oculomotor paralysis of one side with tremor of the opposite arm and leg, this is known as *Benedikt's syndrome*. Moreover, certain lesions of the optic thalamus cause loss of emotional mobility in the opposite side of the face, with little or no weakness on voluntary movement.

Let us now pass to the irregular, non-rhythmic, spontaneous movements. Of these **chorea** furnishes one of the most striking examples. All are familiar with the irregular, jerking, wriggling, grimacing movements of a choreic child. They may affect the face, soft palate, tongue, trunk, limbs, and even the muscles of respiration. The movements of the limbs are often more marked on one side than on the other, and may be confined to one side—so-called hemi-chorea. Choreic movements occur spontaneously but are increased by emotion or by voluntary movements. They cease during sleep. A good method of eliciting choreic movements in a slight case of the disease, is to make the child hold both hands above the head, when after a few seconds slight involuntary movements appear in the fingers of one or both sides.

The movements of **athetosis** or "mobile spasm" are different, occurring as they do most commonly in the spastic limbs of old hemiplegics (chiefly after infantile hemiplegia). Athetosis never occurs in a limb which is completely paralysed but only when some degree of voluntary power survives. The movements are usually confined to the upper limb, and consist of very slow, irregular, twisting movements, most marked in the fingers and wrist, but in severe cases affecting the forearm, elbow, shoulder, and even the lower limb, where the most common involuntary movement is a hyper-extension of the great toe. Only in bilateral athetosis (generally, though not always, following diplegia) do the movements affect the face, causing hideous grimacing. In a typical case the hand movements consist of slow flexion, hyper-extension, and spreading out of the fingers, irregularly, one after another. Combined with this there is alternate abduction and opposition of the thumb, with flexion or extension at the wrist, and pronation or supination of the forearm. Figs. 29 to 32 are taken from a well-

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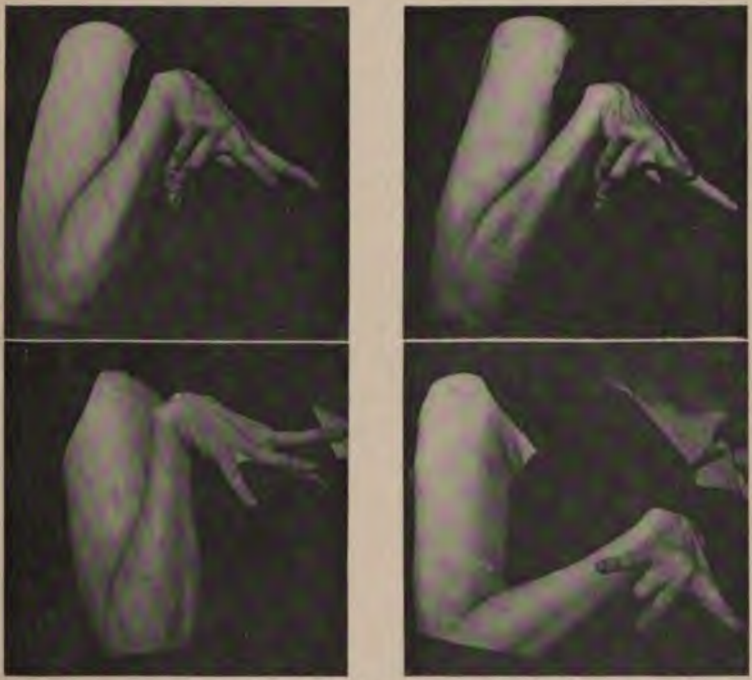
[1] Holmes, *Brain*, 1904, vol. xxvii. p. 327.



FIGS. 29 to 32.—Athetosis in a woman aged 29, the subject of right-sided hemiplegia of nineteen years duration. There was total paralysis of the right upper limb from the shoulder downwards. The figures show athetosis of fingers, wrist, and elbow.

marked case of athetosis in which the movements affected all the joints of the upper limb. Athetosis is intensified by voluntary movement whether of the paralysed limb or of the opposite unaffected hand. It cannot be controlled by voluntary effort, and sometimes persists even during sleep.

Many healthy people make automatic extension movements of the upper limbs during the act of yawning. By the ancients these were termed **pandiculation.** Such movements when the patient yawns or is tickled are often well seen in the paralysed limbs of a case of severe hemiplegia, and the patient may harbour false hopes of a return of motor power by observing extension movements of his paralysed fingers or elevation of the paralysed arm. Unfortunately these movements are not a hopeful sign in hemiplegia; on the contrary, the more severe the lesion of the pyramidal tract, the more marked is the pandiculation. It does not occur in limbs which are the subjects of athetosis or other involuntary movements. Pandiculation has been ascribed by Bertolotti[1] to irritation of the thalamic centres.

Spontaneous movements also occur in cases of advanced **Friedreich's ataxia,** being most marked in the head, neck, and face. They commonly consist in irregular nodding movements of the head or grimacing, which has been called "nystagmus of the face." But in Friedreich's ataxia the most outstanding feature is inco-ordination of voluntary movements. The absence of the knee-jerks, the deformity of the feet, the scoliosis, &c., will all point to a correct diagnosis.

Again, in **general paralysis of the insane,** spontaneous tremors of an irregular type are frequently observed, even at rest. They occur chiefly in the face, especially if the patient be emotionally excited or just about to speak. Waves of fibrillary tremor appear, rippling along the muscles of the lips, tongue, and face. Here we have other physical signs to guide us, *e.g.* the slurring articulation, the irregular or unequal pupils, frequently of Argyll-Robertson type, exaggeration or loss of the knee-jerks, the condition of the cerebro-spinal fluid, and the characteristic mental symptoms.

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[1] *Revue Neurologique,* 1905, p. 953.

There is another great group of involuntary movements which includes the habit spasms, the tics, and the reflex spasms. A proper classification of these is well-nigh impossible, since the different varieties merge into each other. All are most common in people of "nervous" constitution, all are increased by emotion, and they cease during sleep. Slight degrees of **habit-spasm** may occur without any other sign of functional disease. Public speakers, such as clergymen, barristers, actors, and even medical lecturers, occasionally have little unconscious "tricks" of movement. A distinguished university professor has frequent clonic jerks of the frontales muscles, which suddenly elevate his eyebrows; a popular comedian makes rapid blinking movements of both eyes (blepharo-spasm) when he advances towards the foot-lights; a young lady has clonic spasms of both platysmata when her neck is exposed in evening dress at dinner parties, and so on. Such minor degrees of habit-spasm are usually bilateral and occur without any local exciting cause.

More severe varieties of habit spasm are included under the term "**Tic.**" A tic is of cortical, not reflex origin, and consists in the frequent explosive repetition of the same motor act—generally a violent, irregular one, such as rapid shaking or tossing of the head, grimacing, wriggling of the shoulder, &c. It passes off as suddenly as it comes on. Moreover, it does not interfere with voluntary movements. For example, however violent a tic of the right shoulder or arm may be, the patient's handwriting shows no abnormality. As Patrick[1] has put it, when the impulse to tic can no longer be controlled, the patient takes pen from paper, executes his tic and then resumes writing. At first sight, tic might be confounded with chorea. But chorea does not repeat the same movement regularly again and again. Patients who have severe tic usually show signs of mental degeneracy, they are emotional and psychically childish, and are often the subjects of various forms of "phobia." The greater the psychical abnormality, the more inveterate is the tic. "Tiqueurs" often have explosive articulation, "word-swallowing," sudden stoppage of

[1] *Journal of American Med. Assoc.*, Feb. 21, 1905.

speech, disordered respiration, echolalia (repetition of a particular word or phrase), or coprolalia (repetition of a blasphemous or obscene word).

Spasmodic torticollis is one of the commonest and most severe varieties of tic. Though the movement of the head is apparently a unilateral one, the head being jerked constantly to one side, commonly to the left (see Fig. 33), it is really bilateral, since muscles on both sides of the neck are employed to produce the movement, and the movement is sometimes combined with a backward jerk, a so-called retro-collic spasm. Severe torticollis usually begins after middle life. At first occurring in paroxysms, it ultimately becomes continuous during waking hours, and the affected muscles become hypertrophied from over-use.

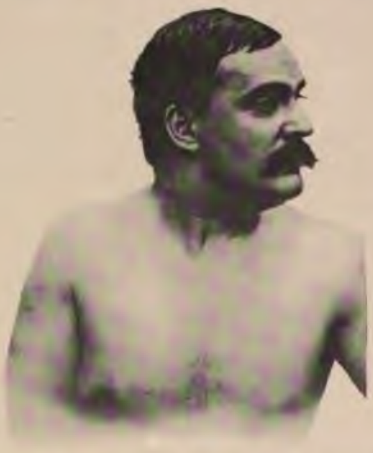

FIG. 33.—Spasmodic torticollis, with secondary hypertrophy of right sterno-mastoid.

There is another group of movements which, originally excited by some peripheral irritation, may be classed as **reflex spasms.** A spasm, unlike a tic, begins locally, perhaps in a single muscle, and spreads to adjacent muscles. When the exciting stimulus is unilateral, the reflex spasm is usually unilateral also, but not invariably so, for bilateral reflex spasms also occur, as in tonic and clonic contraction of the orbiculares oculorum (blepharospasm), the result of corneal or conjunctival inflammation, or as in the case where a vaginal caruncle or anal fissure produces vaginismus with bilateral adductor spasm. Of the unilateral reflex spasms one of the most striking is the intense facial spasm which occurs in severe cases of trigeminal neuralgia or *tic douloureux*, which is not a true tic but a reflex spasm. In this disease the patient has paroxysms of agonising pain in one or more divisions of the trigeminal nerve. During a paroxysm, the face on the side of the pain is thrown into strong tonic contraction, the eye is closed, the mouth is drawn up on the affected side, and the patient often presses his hand desperately over the site of pain. Only when the acute stage of the paroxysm passes off do the facial muscles relax. Less severe unilateral *facial spasm*, either tonic or clonic, implicating some or all of the facial muscles, may occur from other reflex causes, generally in the territory of the fifth nerve, such as a decayed tooth, a non-erupted wisdom-tooth, a nasal polypus, &c. But unilateral facial spasm also occurs, less frequently, in lesions of the facial nerve itself, as for example when tumours or abscesses compress the nerve. Sometimes it follows an attack of ordinary facial palsy, less commonly it may precede its onset, so that in every case of unilateral facial spasm we should search for local lesions, not only of the trigeminal but also of the facial nerve.

Sometimes a reflex-spasm may persist as a habit-spasm, long after the original exciting cause has passed away. Such cases can usually be diagnosed by their history. Thus a lad lost his left arm by avulsion in a machinery accident. The stump was amputated at the shoulder-joint, but clonic spasms appeared in the trapezius and scapular muscles, and these persisted after all the posterior nerve-roots in that region were divided by operation. But other cases occur, even of unilateral spasm, without any reflex exciting cause or the history of one, and they are difficult to classify. Thus a lady whose menopause occurred at the age of forty-five, at the same time also lost most of her property through the failure of a bank. She gradually developed clonic spasm of the left facial muscles. At first, this consisted merely in a slight flickering of the lower lid for a second or two, every few days, but the condition gradually increased in severity until, when she came under observation thirteen years later, the spasms affected all the facial muscles on one side, beginning as a flickering movement, and then becoming tonic and lasting from twenty to thirty seconds at a time, the eye being closed, the eyebrow elevated, the angle of the mouth drawn outwards, and the platysma thrown into strong contraction. In the intervals between attacks the face was symmetrical. Under treatment by bromides and galvanism this case became rapidly better.

Finally, there are numberless varieties of **hysterical spasms,** apart from the hysterical "fits," which have already been discussed. We can only refer to some of the commoner types. Thus *saltatory spasm* consists of a series of jumping or skipping movements, which occur whenever the patient assumes the erect posture. A similar spasm, less severe in degree, may produce paroxysms of trembling in the legs, as in a hysterical girl of nineteen with many other stigmata of hysteria, in whom the spasms ceased at once when she lay down. All sorts of movement, however, may occur in hysteria, simulating almost any kind of tremor. For instance, another hysterical woman of twenty-one had constant movements of the face, left arm, and both legs, resembling those of athetosis but being very much faster. In her case typical hysterical contractures and segmental anæsthesia of the hysterical type, together with the normal state of the reflexes, &c., aided in the diagnosis of hysteria, which disease will be further considered in a subsequent lecture.

LECTURE VII

APHASIA

We exchange ideas with our fellow-men chiefly by means of speech. Speech is an arbitrary code of signals, vocal or written. These signals are perceived by our auditory or visual centres. Every country has its own particular code or language, which is learned by each of its inhabitants. Gestures and mimic movements, as a means of communication, although international, have a very limited field of usefulness as compared with speech. Two individuals, each ignorant of the other's language, can certainly communicate with each other after a fashion by means of gestures alone, but they cannot express many ideas in this way, but only simple primitive emotions such as pleasure, anger, surprise, and so on, or pantomimic imitations of certain acts.

There are three chief classes of cases in which the functions of articulate speech may be lost. Firstly, there are conditions in which the patient's higher intellectual functions are in abeyance, either congenitally as in idiots, or from disease as in acquired dementia, coma, or in certain cases of hysteria. Such patients are speechless, but they are not aphasics. Secondly, there are the cases where the higher intellectual centres are capable of function, but the cortical speech-centres which control the motor acts of speaking and writing, or the sensory processes of recognising spoken or written words, are diseased, and yet the patient has not necessarily any paralysis of the peripheral organs of speech, nor is he necessarily deaf or blind. To this group the term "aphasia" is applied. Lastly, there are the cases where, with intact intellectual functions and normal cortical speech-centres, there are defects in the peripheral organs of articulation, so that the patient is unable to articulate distinctly—for example, cases of cleft palate, post-diphtheritic palsy of the palate, facial or hypoglossal paralysis, bulbar paralysis, and so on. These are affections, not of speech proper, but of articulation.

Aphasia may be defined as impairment or loss of speech due to the loss of memory for those signs, vocal or written, by means of which we exchange ideas with our fellow-men. An aphasic, unless his higher intellectual centres are impaired, usually preserves his powers of gesture and of pantomime. Aphasia is due to disease, organic or functional, of certain well-defined special centres in or near the cortex of the brain. These cortical centres exist on both sides of the brain, but ordinarily in right-handed people the speech-centres on the left side of the brain are predominant.

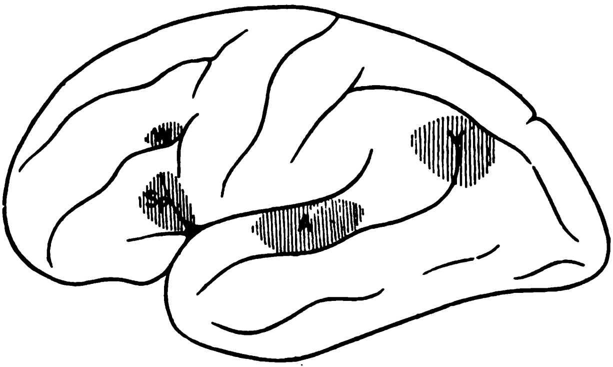

FIG. 34.—Diagram of left cerebral hemisphere, showing approximate positions of the centres concerned in speech.

A.	Auditory word-centre.	Sensory.
V.	Visual word-centre	
Sp.	Vocal word-centre.	Motor.
W.	Motor centre for writing.	

Let us consider the speech-centres somewhat more in detail. For the interchange of ideas two distinct processes are required—one sensory, the other motor. The sensory process includes the hearing and understanding of spoken words, and also the seeing and understanding of written or printed letters. The memories of words heard and seen are stored up in specialised parts of the auditory and visual centres, named respectively the auditory word-centre and the visual word-centre (Fig. 34). The auditory word centre is at the posterior end of the first temporo-sphenoidal convolution, the visual word-centre is in the angular gyrus. Either centre may be diseased; so that we have two varieties of sensory aphasia, viz.—**auditory aphasia** and **visual aphasia.** Then there is in speech the motor element, consisting of the motor act of expressing ourselves in words, either vocally or by means of writing. The memories of these motor acts of vocal speech are stored up at the posterior end of the inferior frontal (Broca's) convolution, and the adjacent part of the ascending frontal convolution. If this centre be destroyed, we have **motor aphasia** or **aphemia.** Earlier writers used to describe a separate centre for writing (independent of the vocal word-centre), a lesion of which would produce loss of the faculty of writing—**agraphia.** But no case has been verified pathologically in which a focal lesion has produced pure agraphia without affection of vocal speech, so that the writing-centre, although it must be represented diagrammatically in any theoretical scheme of cortical speech-centres, is probably merely a part of the ordinary psycho-motor centre for the upper extremity.

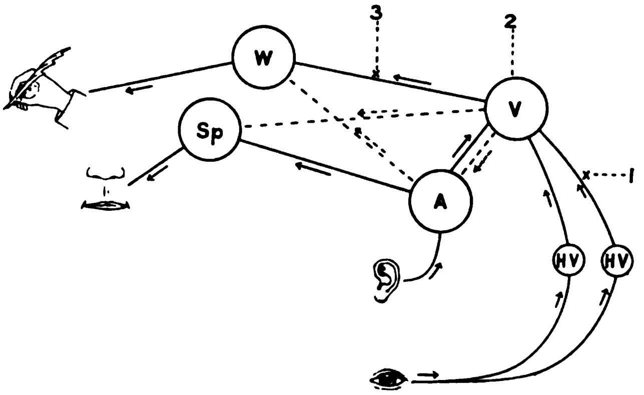

FIG. 35.—Diagram of Speech-Centres (after Bramwell).

A. Auditory word-centre. V. Visual word-centre.
Sp. Motor vocal word-centre. W. Motor centre for writing.
HV. Half-vision centre.

The interrupted lines indicate possible but less habitual routes for transmission of impulses.

Fig. 35 is a scheme of the connection of the various centres concerned in speech. Let us first notice that the motor vocal word-centre is subservient to the auditory word-centre, and that the writing-centre is similarly subservient to the visual word-centre. A child learns to speak first by hearing spoken words and then imitating them. Therefore speech at first is entirely auditory in origin. Later, in learning to read, the meaning of each word is learned by associating the letters seen with words heard spoken, so that the auditory word-centre acts as the instructor of the visual word-centre.

In most people, during the process of silent thought, words are revived primarily in the auditory word-centre, and there is usually a simultaneous revival of the same words in the visual word-centre. But in other people the revival in the visual word-centre comes to be of greater importance. Accordingly we may classify people into "auditives" and "visuals" according to their mode of revival of words in thought. Most of us are "auditives." Simultaneous revival of word-images in several speech-centres makes our comprehension of the idea more perfect. Thus a difficult concept is better understood if we read it aloud, because this involves the activity of the visual, auditory and vocal word-centres.

Aphasia commonly results from organic disease of one or more of the cortical speech-centres, or of the sub-cortical fibres connecting them. The most frequent organic causes are embolism, hæmorrhage, or thrombosis, cerebral abscesses and cerebral tumours. The differential diagnosis between these various conditions depends largely on the history; embolism producing the symptoms suddenly, hæmorrhage taking several minutes, thrombosis taking perhaps hours, abscess and tumours being still more gradual in onset. But we also meet with cases of temporary or functional aphasia, sometimes from mere debility or exhaustion, sometimes following a paroxysm of migraine or a "congestive attack" in general paralysis, or after an epileptic fit.

In investigating a case of aphasia we should first note whether the patient has other signs of gross cerebral lesion, such as hemianopia, or hemiplegia, and should inquire whether he is naturally right- or left-handed. Most children are taught to write with the right hand, whether they are right-handed or not, and therefore in determining this point we inquire with which hand a man draws a cork, throws a stone, &c.; or if a woman, with which hand she combs her hair.

The following series of inquiries (based on Beevor's scheme) should then be made. The capital letters in parenthesis indicate the parts of the brain involved in each case.

1. Can the patient spontaneously utter intelligible words? (**Sp.**) Note the extent of his vocabulary. Can he pronounce all words or only a few? Get the patient to talk spontaneously, and observe whether he talks fluently or misplaces words or syllables, whether he talks in disjointed phrases, ("telegraphic" type of speech,) or whether he talks unintelligible jargon.

2. Can he understand words which he hears? (**A.**) Ask him to touch his nose, ear, eye, chin, &c., in turn, thus testing his interpretation of nouns. Then ask him to smile, whistle, shut his eyes, &c., thus testing his comprehension of verbs. Sometimes we find that the patient executes the first command correctly, but continues to repeat the same act in response to different commands. A patient can sometimes sing the words and air of a song, when he is unable to repeat the words in a speaking voice.

3. Can he understand written questions or commands which he sees? (**V.**) Write down and show him simple sentences, such as "How old are you?" "Put out your tongue." "Give me your left hand."

4. Can he write spontaneously? (**W.**) If his right hand is paralysed, let him try with the left. Observe whether he writes intelligibly, whether he misplaces words or syllables, or whether he scribbles meaningless signs.

5. Can he copy from printed to written letters? (**V→W.**) Print some word such as "Hospital" or "Monday," and get him to copy this.

6. Can he write to dictation words which he hears? (**A→V→W.**)

7. Can he pick out objects of which he hears the name? (**A→V**) Place in front of him a heap of objects, such as a key, a shilling, a match, a pencil, and ask him to pick out each in turn.

8. Can he repeat words heard? (**A→Sp.**) Try him first with

simple words and phrases; *e.g.* "cat," "dog," "nurse," "good-morning," &c.

9. Can he name objects seen, and can he read aloud from words shown to him? (**V→A→Sp.**) Point to different objects and ask him what they are.

10. Does he understand gestures and pantomimic movements? Without speaking to him, get him to imitate you when touching the nose, spreading out the fingers, protruding the tongue, &c.

Auditory Aphasia, or Word-Deafness.—The patient in this case is not deaf, but simply word-deaf. He hears ordinary sounds and noises, but spoken words are not understood; they sound to him like an unknown tongue. The character of the symptoms varies according as the lesion is subcortical or cortical in position.

(*a*) **Subcortical, or Pure Auditory Aphasia.**—This is extremely rare. Here the lesion simply blocks the way-in for spoken words The patient therefore has word-deafness—*i.e.* he is unable to understand what is said to him; he is also unable to repeat spoken words or to write from dictation. But the auditory word-centre being still intact, he possesses all his memories of auditory speech, and therefore spontaneous speech is perfect. Moreover, the visual word-centre being in normal working order, he is still able to read, and, as a matter of fact, reading is his only means of eceiving messages from other people.

(*b*) **Cortical Word-Deafness.**—This is much commoner than the other variety. Here the lesion involves the cortical centre itself, and the auditory memories of spoken words are obliterated. And therefore, in addition to the previous defects of word-deafness with inability to repeat spoken words or to write from dictation, there are other symptoms due to the fact that the motor speech-centre is no longer controlled by the auditory word-centre. Internal speech and thought are impaired, and so the patient makes mistakes whether in speaking spontaneously or in reading aloud. He also makes mistakes in writing, especially in spelling. He talks fluently enough, it is true, but he tends to mix up his words or syllables, and in a severe case may jabber unintelligible jargon. Word-deafness renders the patient unaware of his own errors. This, as we shall see, is in marked contrast with motor aphasia, where the patient recognises his own mistakes as soon as he has uttered them. If the lesion of the auditory word-centre be incomplete, the word-deafness and resulting errors of speech are also partial. These latter may, in a slight case, be confined to inability to name objects, *i.e* nouns, the patient being still able to express abstract ideas. Thus a partially word-deaf patient, who is unable to name a knife shown him, may say, " It is for cutting." Or again, partial word deafness may produce simply confusion of words; the patient may say one word when he means another (*paraphasia*). It rarely happens that word-deafness remains permanent and complete; the auditory word-centre in the opposite hemisphere generally compensates, to some extent, as time goes on.

The extent of mental disturbance in word-deafness varies according to whether the patient be a strong " auditive " or a strong " visual." In the latter case the mental impairment is much less than in the former, and the disturbances of motor speech are but slight.

The auditory and visual word-centres are fairly close together, and more than this, they are supplied by the same branch of the middle cerebral artery (see Fig. 19, p. 34); so that it is not uncommon for a single arterial lesion, *e.g.* a thrombosis, to affect both centres together and to produce a combination of word-deafness and word-blindness.

Visual Aphasia, or Word-Blindness (Alexia).—In word-blindness the patient can see, but cannot understand printed or written characters. They appear to him like strange hieroglyphics. He sees the shape of the letters, but they convey no meaning to his mind.

Here, as in word-deafness, the symptoms vary in degree. The patient may be unable to recognise a single letter (letter-blindness), or he may be able to spell out the letters singly but unable to read syllables or words. Often a patient who is unable to read any other word, can recognise his own name. Frequently he retains the power of recognising numbers and of doing addition, subtraction, and multiplication sums. We have to recognise two varieties of word-blindness, according as the lesion is cortical or subcortical in position.

(*a*) **Subcortical, or Pure Word-Blindness.**—Here the way-in for visual word-impression is destroyed, the visual word-centre remaining intact. The patient cannot understand written or printed words, nor can he read aloud, but he still retains the power of writing spontaneously and from dictation; and therefore he can express his thoughts perfectly in writing, but is unable to read what he has himself written. Inasmuch as a subcortical lesion, in order to produce word-blindness, must be beneath the angular gyrus and in such a position as to cut off the afferent visual impulses from both half-vision centres to the visual word-centre in the angular gyrus, it will necessarily implicate the optic radiation to some extent. And therefore a pure or subcortical word-blindness is always associated with hemianopia or, when partial, with hemi-achromatopsia (Fig. 36).

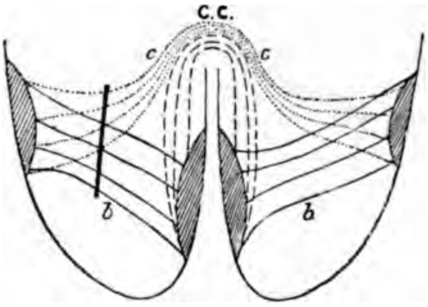

FIG. 36.—Diagram representing a lesion (indicated by thick black line) which produces pure word-blindness (Bastian).

C. C. Posterior extremity of corpus callosum.
c. c. Commissural fibres connecting the two visual word-centres.
b. b. Fibres connecting each half-vision centre with the visual word-centre of the same side.

(*b*) **Cortical Word-Blindness.**—In this variety, in addition to the symptoms just described, there is loss of the memories of written or printed words, and therefore, inasmuch as the visual word-centre, which controls the writing-centre, is destroyed, the patient has **agraphia.** He is unable to write spontaneously, to copy from printed into written characters, or to write from dictation. If the lesion of the visual word-centre be partial the alexia and agraphia are also incomplete, and mistakes in writing may amount simply to the writing of wrong words, syllables or letters—so-called "*paragraphia.*"

These points are illustrated by Bramwell's well-known diagram (Fig. 35), in which a lesion at the position marked 1, destroying fibres passing from one half-vision centre to the visual speech centre, would not produce word-blindness. A lesion at 1, destroying fibres passing from both half-vision centres to the visual speech centre, would produce word-blindness but not agraphia, since the visual speech centre is unaffected and is able to influence the motor writing-centre. A lesion at 2, destroying the visual speech-centre, would produce word-blindness and agraphia. A lesion at 3 would produce agraphia (unless the motor writing-centre could be brought into action in some roundabout way), but not word-blindness, the visual speech-centre being intact.

Motor Aphasia.—In this variety the patient has lost the power of expressing himself by spoken words. He can neither speak spontaneously nor can he read aloud. And yet (unless the motor speech-centres on both sides of the brain are destroyed) he is not absolutely dumb. As Hughlings Jackson puts it, the patient is speechless, but as a rule not wordless. He can usually utter intelligently a few words, such as "yes" and "no," by means of the speech-centre on the uninjured side, and in addition he may have other words or phrases, mostly interjections, such as "oh!" "oh my!" "damn!" "by Jove!"—so-called "recurrent utterances"—which he utters automatically when excited, or when making an effort to speak. A patient who has motor aphasia, unlike a word-deaf person, is conscious of his own errors.

We have to distinguish two varieties of motor aphasia, according as the lesion is cortical or subcortical.

(*a*) **Subcortical, or Pure Motor Aphasia.**—Here the patient, though perfectly intelligent and able to understand spoken and written language (by means of his uninjured auditory and visual word-centres), cannot utter spoken language, either spontaneously by reading aloud or by repeating what he hears. This is because the way-out for spoken speech is blocked. But his mental speech processes are unimpaired, and if the outgoing fibres from the writing centre are unimpaired, he is able to express himself in writing, as in the scriptural case of Zacharias, the father of John the Baptist (St. Luke, chap. i.), and in many cases the patient can indicate by signs how many syllables or letters are in the word which he desires to speak but cannot utter.

(*b*) **True Cortical Motor Aphasia.**—In this the cortical centre of Broca is destroyed. The patient has not only all those defects of a subcortical case, which we have just considered, but, in addition, his mental processes of internal thought are impaired, since the memories of words spoken and written by himself are impaired. He has therefore difficulty in understanding complicated sentences, whether spoken or written. Together with difficulty in vocal speech there is often (though not always) associated a similar difficulty in writing—*agraphia*—proportional to the defect of speech.

Pure isolated agraphia, without any other symptom, does not occur, and as we have seen, the commonest variety of agraphia is that due to cortical word-blindness. The study of agraphia due to sensory aphasia is easier than that associated with motor aphasia, because in the sensory aphasia there is no necessary motor paralysis of the right arm or hand, whereas in motor aphasia the patient has to make his attempts at writing with the left hand.

Such are the chief types of aphasia. Clinically, however, it is commoner to meet, not with pure auditory, visual, or motor aphasia, due to a small focal lesion, but with combinations of these, or with total aphasia, the result of a larger destructive lesion implicating several or all of the speech centres. Such severe aphasias, of course, produce a more profound degree of mental deficiency, and inasmuch as the same artery, the middle cerebral, supplies not only the speech centres, but also the other cortical motor areas and the corpus striatum (see Fig. 19), total aphasia is usually combined with severe right hemiplegia.

LECTURE VIII

DISORDERS OF ARTICULATION

It is necessary to distinguish clearly between speech and articulation. Speech is a cortical function, articulation is mainly bulbar.

Disordered articulation, or **dysarthria**, signifies difficulty in performing the co-ordinated muscular movements necessary for the production of the consonants and vowels which go to form syllables and words.

In simple dysarthria there is no affection of the cortical centres or paths which are concerned in the processes of speech proper. A patient, for example, who has advanced bulbar palsy, even when he is unable to articulate a single word, is not, strictly speaking, speechless. He is inarticulate, which is quite a different thing. His mental speech-processes remain normal, he can still express himself fluently in written speech, and he is able to understand everything he hears or sees.

The peripheral mechanism of vocal speech is partly musical (or voiced), consisting in vibration of the approximated vocal cords. It is also partly consonantal (or voiceless), consisting in the co-ordinated action of numerous muscles of the lips, tongue, palate, and pharynx. The term "articulation" is specially applied to this latter, the consonantal element.

To test a patient's power of articulation, we listen to his ordinary conversation, or we ask him to read aloud a passage from a book or newspaper, and then notice how he pronounces his words. Then, if he shows any abnormality in connection with particular consonants, we ask him to repeat "catch" words, chosen so as to present special difficulties, *e.g.* "British constitution," "biblical criticism," "West Register Street," "hippopotamus," "Burgess's fish-sauce shop," &c.

Difficulties of articulation are the result of defects, consisting either in paralysis or inco-ordination, of certain groups of muscles in the lips, tongue, palate, pharynx, or larynx. And the underlying defect may be either in the bulbo-muscular neurones (comprising the pontine and medullary nuclei, the peripheral motor nerves and the muscles), or it may be due to supra-nuclear affections in the cortico-bulbar neurones, at some point between the higher speech-centres and the bulbar nuclei. Or it may be due to ataxia of the organs of speech.

Infra-nuclear and Nuclear Affections of Articulation.—Articulation may be impaired as a result of paralysis of any of the motor nerves or nuclei supplying the articulative muscles.

Unilateral hypoglossal paralysis (as in the case of a business man shown in Fig. 65, in whom this nerve was divided by a stab in the neck from a discharged employé) produces motor paralysis followed by atrophy of the corresponding half of the tongue. The paralysed half-tongue feels to the patient as if he had a foreign body in his mouth. This makes his articulation clumsy, lisping and indistinct, especially in the case of linguo-dental (S, Z, Th.) and anterior linguo-palatal consonants (T, D, L, R.). But after a few days the patient becomes accustomed to the feeling of his palsied half-tongue, and his articulative difficulty to a large extent disappears.

Facial palsy, from its affection of the lips, renders labials (P, B.) and labio-dentals (F, V.) indistinct, and especially so when the palsy is bilateral.

Bilateral paralysis of the palate, *e.g.* post-diphtheritic paralysis, congenital cleft palate, and syphilitic perforation of the palate, all produce the same articulative difficulty, inasmuch as the nasal cavity cannot be shut off from the mouth. As a result the voice is nasal, and certain consonants are altered (B becomes M, D becomes N, K becomes Ng, and so on), so that articulation as a whole is indistinct.

Unilateral paralysis of the recurrent laryngeal nerve renders the voice hoarse, by paralysing one vocal cord, and so interfering with the phonation of vowels. But it does not interfere with the articulation of consonants. If the vagus root be affected at its point of exit from the medulla, the soft palate is often paralysed on the same side (see later, Cranial Nerves). But unilateral palatal palsy, unlike bilateral, does not affect the articulation of consonants.

Articulation may also become indistinct from disease of the bulbar nuclei—so-called glosso-labio-laryngeal paralysis, or *bulbar palsy*. In this disease there is a diffuse, progressive weakness of the muscles of articulation, with atrophy and fibrillary tremors of the muscle fibres, especially those of the tongue and lips. Articulation becomes more and more indistinct, saliva dribbles from the patient's trembling lips, and in advanced cases there is interference with swallowing and coughing. If, as is often the case, bulbar palsy is an upward extension of an amyotrophic lateral sclerosis, there may be a concomitant muscular atrophy and fibrillary tremor in the muscles of the upper limbs, especially in the intrinsic hand muscles. And, owing to affection of the pyramidal tracts, there is also exaggeration of the deep reflexes.

There is a form of *myopathy*—the *facio-scapulo-humeral type* of Landouzy and Dejerine, in which the facial muscles are atrophied. This affection is a bilateral one and the patient's lower lip protrudes in a characteristic fashion which has been termed the "tapir" type of lip. He has also a peculiar "transverse" smile. In severe cases of this disease the articulation of labial consonants becomes impaired just as it does in double facial palsy.

Myasthenia gravis, when it affects the bulbar muscles, reproduces all the features of bulbar palsy, with this difference, that the paralysis varies in its degree from time to time, becoming accentuated by fatigue. After a period of rest a patient so affected may resume with normal articulation, but, as he continues to speak, his muscles gradually become exhausted, and his articulation becomes more and more indistinct. Not only the lips and tongue, but the ocular muscles, the muscles of mastication and various muscles of the trunk and limbs, show evidence of temporary paralysis or fatigue, and ultimately the patient succumbs to fatigue of his respiratory muscles.

Articulative Ataxia.—There are some diseases in which articulation becomes indistinct, not from paralysis of the muscles but from ataxia.

In *Friedreich's ataxia*, for example, the articulation becomes slow, thick, and clumsy, and the patient talks as if he had a foreign body in his mouth, so that his speech has been aptly termed the "hot-potato" speech. The pitch of the voice in this disease may vary from word to word, and in advanced cases a certain degree of mental feebleness is often superadded.

In laryngeal *tabes* where the larynx is affected by ataxy the voice is tremulous, and when a tabetic patient has ataxia of the tongue his articulation becomes laboured. This lingual trouble in tabes is often associated with a peculiar constant rolling movement of the tongue on the floor of the mouth, even when the patient is not speaking. And the patient often has an annoying subjective sensation in his tongue, as if it were covered with blotting-paper, which he tries to get rid of by a restless rolling of the tongue.

Supra-nuclear or Cortico-bulbar Affections of Articulation.—Articulation may be affected where the upper or cortico-nuclear neurones are affected. The slurring articulation of many cases of acute *alcoholic intoxication* is familiar to laymen, being especially marked in the pronunciation of labial and anterior linguo-dental consonants. In rare instances alcoholic dysarthria persists for days after the alcoholic celebration. Tollmer and other French authorities ascribe this circumstance to cerebellar intoxication. Many alcoholic patients realise their own articulative difficulty, and in endeavouring to compensate for it, they utter certain words with a deliberateness and undue emphasis that betrays them.

Very similar to the alcoholic articulation is that of *general paralysis of the insane*. But the typical general paralytic shows also a characteristic fibrillary tremor of all the muscles around the lips and nose.

Both in acute alcoholism and in general paralysis there is often a tendency to choose an occasional wrong word, or to misplace syllables. Such defects are not bulbar but cortical in origin.

In the later stages of general paralysis, articulation may be totally unintelligible, being reduced to a mere mumble.

After an attack of hemiplegia, right-sided, the patient is often aphasic. But even in a left-sided hemiplegia where no true aphasia exists, it is not uncommon to find a temporary change in the articulation, which loses its crispness, and becomes a little laboured and indistinct. This condition usually passes off after a few days.

In cases where a patient has a bilateral hemiplegia we often meet with "*pseudo-bulbar*" *paralysis*. The most common history is that there have been one or more hemiplegic attacks, all confined to the same side, but at last the patient has an attack on the opposite side. This now produces, in addition to the classic signs of a double hemiplegia, pseudo-bulbar paralysis, with thick, indistinct articulation, closely resembling that of true bulbar palsy and with the same difficulty in swallowing, coughing, &c., but without atrophy or fibrillary tremors of the affected muscles. The pseudo-bulbar patient is emotionally irritable; he laughs, or more often cries, on slight provocation, and, unlike the subject of true bulbar paralysis, he is generally somewhat deficient mentally. And there is usually a history of successive (more rarely of simultaneous) attacks of hemiplegia on opposite sides of the body.

Double athetosis is a disease which is usually congenital. It is characterised by wild wriggling and twisting movements of all the limbs, chiefly on attempted voluntary movements, and by grimacing of the face, together with spastic rigidity of the affected muscles. There is generally a certain degree of mental deficiency. In this disease articulation is often affected. The grimaces of the face and of the tongue muscles interfere considerably with articulation. Moreover, irregular spasmodic contractions of the diaphragm and other respiratory muscles give the voice a curiously jerky or groaning character, due to sudden interruptions of breathing.

Patients suffering from *disseminated sclerosis* frequently have a peculiar so-called "staccato" speech, in which the words are enunciated in a jerky mincing fashion, very difficult to describe, but easy to recognise once it has been heard. This is sometimes called the "scanning" speech, from its fancied resemblance to the scansion of Latin or Greek verse.

In *paralysis agitans*, as the disease advances, the patient's voice becomes thin, feeble and reduced almost to a monotone, whilst his articulation, like his gait, acquires a "festinant" character. When speaking, he begins slowly, but towards the ends of sentences or long words he tends to hurry, so that the final syllables are pronounced hastily. Together with this we have the characteristic "starched," expressionless face, the Parkinsonian mask, commencing unilaterally and ultimately becoming bilateral.

Severe cases of *chorea* may have the articulation severely interfered with, owing to sudden violent movements of the face, tongue, and respiratory muscles. Speech becomes hesitating and jerky, and in very bad cases the voice may be reduced to a whisper.

There are also affections of articulation due to functional cortical disturbances. Of these the most familiar is **stammering,** which consists in a want of co-ordination between the vocal (laryngeal) and consonantal (oral) mechanisms of speech, so that, in the common type, the patient sticks at a consonant, which he often continues to repeat, over and over again. He misdirects his energy on the consonants, instead of touching them lightly and passing on to the vowel sounds. Most stammerers lose their stammer when they sing, their attention being then directed to the vocal part of speech.

A rarer variety of stammering is that in which the patient sticks at initial vowels. This is due to temporary spasm of the false vocal cords, and the patient remains with his mouth wide open until the spasm relaxes, when his words suddenly rush forth in a hurried stream until he has no breath left. He then takes another breath and the precipitate rush again occurs.[1]

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[1] Cathcart has pointed out that this variety of stammering is described in Shakespeare's "As You Like It" (Act iii. Scene 2) as follows:—

"I would thou couldst stammer, that thou mightst pour out this concealed man out of thy mouth, as wine comes out of a narrow-necked bottle, either too much at once, or none at all. I prithee, take the cork out of thy mouth that I may drink thy tidings."

Many stammerers acquire various tricks, chiefly through their efforts to overcome the stammer. Thus extra noises may be thrown in, *e.g.* sudden inspiratory grunting or whooping noises, or they make grimaces or curious contortions of the limbs.

This leads us to recall the various *articulative tics* or habit-spasms which are met with, either in conjunction with stammering or independently of it, and these may be of the most varied character. The patient's speech may be interrupted by weird pharyngeal barking or grunting noises. Or the articulation may be monosyllabic, a whole breath being taken for each syllable. Or, again, it may be jumbled up in the most extraordinary ways, though hysterics usually interpolate, now and then, a clearly articulated sentence or phrase amongst the other unintelligible ones. Hysterical aphonia is fairly common, and can usually be recognised by the history, together with the characteristic laryngeal appearances. We also meet with cases of hysterical mutism, where the patient refuses to utter even a whispered word.

Deaf-mutes are the patients who are popularly known as "deaf and dumb." A normal child learns to speak by imitating words which he hears, but if a child be congenitally deaf, he does not learn to speak, but remains deaf and dumb. Moreover, if he is born with normal hearing, but subsequently becomes deaf, *e.g.* from middle ear disease or from meningitis, if this occurs before the age of about six years, he usually loses his power of speech. Deaf-mutes can generally be taught to speak again by the oral or "lip-reading" method, where the child imitates the movements of his teacher's muscles of articulation and also learns to phonate, though usually with a harsh, discordant voice. But even deaf-mutes who have never been taught to speak, usually make noises of some sort or other, often pharyngeal snorts and grunts, or spluttering labial noises, and less frequently laryngeal sounds. They do this especially when excited. This was the case with a deaf-mute who used to make weird snorting noises when playing football. These doubtless helped to smite terror into the hearts of the opposite side. The congenital deaf-mute is usually brighter and more clever than the acquired deaf-mute. Deaf-mutes generally have a wonderful command of gestures and signs.

The dumbness which is present in profound degrees of idiocy is not an articulative difficulty, but a true speech affection due to mal-development of the cortical speech-centres. An imbecile child is speechless because he has no ideas to express; in this respect he differs from the deaf-mute, who is often bright and intelligent.

Certain varieties of deficient articulation are met with in children or in adults who are mentally more or less childish. The condition known as **lalling** consists in a want of precision in the pronunciation of certain consonants. Thus a patient may substitute the uvular R for the ordinary linguo-palatal R, or he may substitute W for R, so that " broken reed " becomes " bwoken weed." Or, again, the patient may substitute Th for S and is then said to *lisp*. These last two varieties are sometimes voluntarily assumed, as a fashionable affectation, by young men not overburdened with brain power. More serious varieties of lalling are where the letter L is replaced by some other consonant, so that " elephant " may be pronounced as " edephant," " esephant," " enephant," " erephant," &c. Still worse is it when the patient has difficulty with K or G, their places being taken by T and D respectively.

As a general rule, it may be stated that lalling on a single consonant does not necessarily indicate defective intellect, whereas lalling on many consonants, if the patient has passed the age of childhood, should arouse the suspicion of mental deficiency, although lalling is a normal stage in the process of learning to speak.

Finally, there is the condition known as **idioglossia**, where from difficulty in pronouncing his consonants a child retains the correct vowels, but substitutes other consonants and seems to speak a new language of his own. In the course of time the child usually completely outgrows this weakness.

LECTURE IX

CRANIAL NERVES

The recognition of cranial nerve palsies is, diagnostically, of the utmost importance, nor is it a matter of such difficulty as is commonly imagined.

First, or Olfactory Nerves.—From the under surface of the olfactory bulb on each side there arise some twenty minute nerves which perforate the cribriform plate of the ethmoid to be distributed to the upper part of the nose. To test the sense of smell, we direct the patient to close his eyes. We then hold aromatic substances, such as oil of cloves, peppermint, or asafœtida, in front of each nostril in turn, closing the other nostril with the finger. Ammonia or acetic acid must not be used to test the sense of smell, since these stimulate the fifth nerve (common sensation), and may produce a pungent sensation in the nose, even when the sense of smell is lost.

Anosmia, or loss of the sense of smell, is sometimes of diagnostic value. It may occur, for example, in congenital absence of the olfactory nerves, in lesions of the olfactory bulb or olfactory tract, in injuries of the anterior fossa of the skull, and when there is atrophy of the olfactory nerves in tabes. It also occurs very often in hysterical hemiplegia and is then associated with diminution of the other special senses on the hemiplegic side. But the value of anosmia as a symptom is lessened by the fact that numerous local nasal conditions also produce loss of smell, *e.g.* nasal polypi or even a simple cold in the head.

Parosmia, or perverted sense of smell, and various subjective *hallucinations of smell* occur not only in mental disease but also in gross lesions of the uncinate gyrus, which is the cortical olfactory centre. But here again local nasal conditions may also cause olfactory sensations, *e.g.* the unpleasant odour perceived by a patient suffering from empyema of the antrum of Highmore, from which horribly offensive pus is escaping into the nose.

Second, or Optic Nerve.—This contains not only visual fibres, but also the afferent fibres for the pupillary reflex.

We have already referred to the course of the visual path from retina to cortex (Fig. 17, p. 32). In testing vision we should determine *visual acuity* by means of test types at a fixed distance, such as six metres. Using Snellen's types, of which the largest should be readable at sixty metres, and the smallest at six metres, we direct the patient to read the letters from above downwards. If his vision is normal he will be able to read the smallest type at six metres. His visual acuity is then represented as $V = \frac{6}{6}$. But if he can only read down as far as the type which ought to be visible at thirty metres, then $V = \frac{6}{30}$. Each eye should be tested separately, the test types being well illuminated and the patient standing with his back to the light. When the visual acuity is much impaired, the patient may not even see the largest type, but can only count fingers at a short distance, or perhaps can only tell light from darkness.

Colour vision is most conveniently tested by means of Holmgren's wools. These are thrown on a table well lit by daylight, and the patient is given one particular test-skein of wool which is kept separate from the heap and told, not to name the colour, but to match it, selecting from the heap of coloured skeins all those which are like the test-skein, whether lighter or darker in shade. The patient is given a pale green test-skein. If his colour vision is normal, he will pick out all the pale greens correctly, but if he is red-green colour-blind he will select a grey or straw-coloured skein. Congenital red-green colour-blindness is the commonest variety. Yellow-blue colour-blindness is rarer. If a patient be totally colour-blind he will confuse with the test-skein all those of similar brightness, no matter what their colour may be.

The size of the *field of vision* in each eye is of great importance and for its accurate measurement a perimeter is required. This, however, is a large and expensive apparatus. For clinical purposes the following method is sufficient, presuming that the physician's own visual fields are normal. The physician sits exactly opposite the patient, about a yard away from him and tests each eye separately. To test the patient's *right* eye direct him to cover up his left and to gaze steadily at the physician's left eye. Meanwhile the physician closes his own right eye and looks steadily at the patient's pupil, watching that the patient's eye does not wander from the fixation point. Then the physician, holding his own left hand in a plane midway between himself and the patient, and beginning almost at arm's-length, brings his hand inwards from the patient's ear towards the middle line, meanwhile moving his own

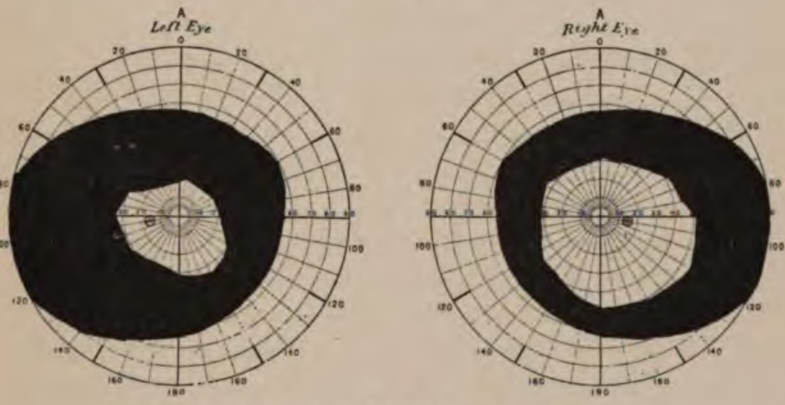

FIG. 37.—Crossed amblyopia, in a case of hysteria.

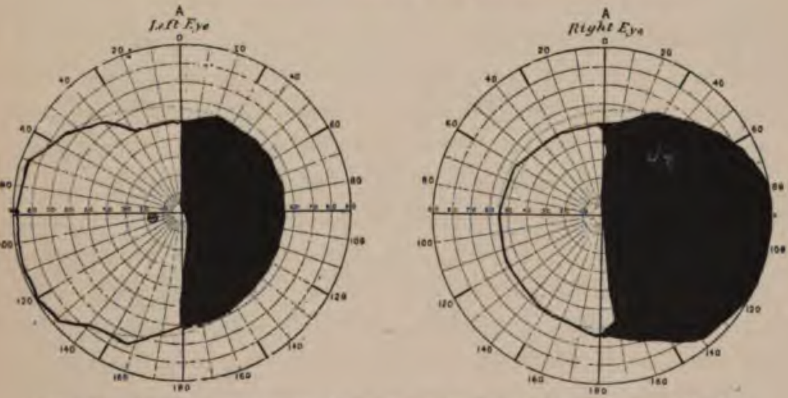

FIG. 37A.—Right homonymous hemianopia, in a case of softening of the left occipital lobe.

fingers. If the patient's visual field is normal, he will catch sight of the moving fingers at the same time that the physician does so. If he does not, that visual field is contracted and the physician then brings his moving fingers inwards until the patient does catch sight of them. In this way we test both the upper and lower quadrants of the field on the temporal and nasal sides, in turn. If we find the visual field diminished in one or other eye, it is well to take a careful perimetric chart.

The visual field may be *concentrically contracted*. This condition is sometimes due to optic atrophy, the field being reduced to a small area surrounding the fixation-point, so that the patient looks at the outer world as though through a narrow tube. More commonly concentric contraction of the visual field occurs in hysteria, the field on the hemiplegic side of the body being more contracted than that on the other side. Less frequently a cortical lesion of the angular gyrus, not implicating the subjacent optic radiation (Fig. 17), causes a similar concentric contraction of both fields, more marked in the eye of the side opposite to that of the brain lesion (Fig. 37). This is somewhat clumsily named *crossed amblyopia*, but, as previously observed, it is much commoner in hysteria than in organic brain disease, and in hysteria it is frequently associated with diminution or loss of other special senses on the side of the more contracted field whose colour sense is frequently lost (achromatopsia).

Hemianopia (Fig. 37A) means blindness of half the visual field, right or left as the case may be, from causes other than retinal disease. It affects the visual field of both eyes, and is due to a lesion of the visual fibres at or behind the *optic chiasma*. Such chiasmic lesions may be the result of pressure by tumours, syphilitic or inflammatory affections of the basi-sphenoid, by tumours of the pituitary body (as in acromegaly), or by tumours of the brain or of its membranes. We have already considered the signs of lesions of the optic tracts, and it is convenient here to recall the effects of **lesions of the optic chiasma.**

(A) If the lesion be in the central part of the chiasma, interrupting the decussating optic fibres (belonging to the nasal halves of both retinæ), there is blindness in the outer half of each visual field:—*bi-temporal hemianopia* (Fig. 38). This sometimes occurs in acromegaly and in the case of other pituitary tumours.

(B) If the lesion be situated at one or other lateral extremity of the chiasma, it will interrupt merely the non-decussating fibres of the optic nerve and optic tract on that side, causing *unilateral nasal hemianopia* in the corresponding eye. To produce bilateral nasal hemianopia there must be two separate lesions, one at each end of the chiasma, a condition which very seldom occurs. A lesion involving the central part of the chiasma and extending to one or other side (Fig. 38, A + B) will produce the sum of these two, namely bi-temporal hemianopia *plus* unilateral nasal hemianopia, *i.e.* total blindness of one eye with temporal hemianopia of the other.

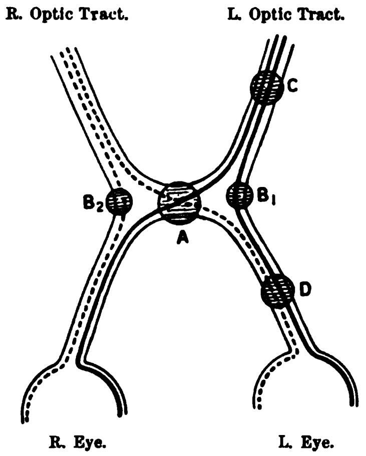

FIG. 38.—Diagram of course of visual fibres in optic chiasma. (Hamilton.)

(C) A lesion of the left optic tract behind the chiasma produces, as already seen, hemianopia in the right halves of both fields of vision.

(D) A lesion of one optic nerve simply causes blindness in the corresponding eye.

In rare cases we may have a *quadrantic hemianopia* in which only one quadrant (instead of one-half) of both visual fields is blind. This is generally due to a lesion limited to part of the cortical half-vision centre in the cuneate lobule. The calcarine fissure divides the cuneus into an upper and a lower part. A lesion of the cuneus above the calcarine fissure causes blindness of the lower quadrant, whilst a lesion below the calcarine fissure causes blindness of the upper quadrant of the corresponding half-field.[1]

The optic discs and retinæ should be examined with the ophthalmoscope in every case of nervous disease. The most important conditions to look for are optic neuritis and optic atrophy. *Optic neuritis* occurs in numerous pathological conditions within the skull, especially in intra-cranial tumours and in tuberculous meningitis. But it also occurs in nephritis, in lead poisoning, in diabetes, and in severe anæmia; and these four conditions must always be excluded before we diagnose gross intra-cranial disease. *Optic atrophy* may occur primarily, as in tabes and disseminated sclerosis, or it may be a secondary post-neuritic process. Apart from optic neuritis and optic atrophy, we must be on the look-out for other pathological conditions of the fundus, such as choroiditis, albuminuric retinitis, tubercle of the choroid, occlusion of the central retinal artery, retinal hæmorrhage, &c.

It must be remembered that a patient may have severe optic neuritis without any impairment of vision. Optic atrophy, on the other hand, causes the visual field to contract concentrically to a greater or less extent, whilst the visual acuity diminishes and ultimately the eye becomes blind.

Third, Fourth, and Sixth Nerves.—It is convenient to study together these three nerves which, between them, innervate all the voluntary muscles of the eye. The distribution of each is as follows :—The *third* nerve (oculo-motorius) supplies all the external ocular muscles except two :—the superior oblique supplied by the fourth nerve, and the external rectus supplied by the sixth. It also supplies the voluntary part of the levator palpebræ superioris (the involuntary part being supplied by the cervical sympathetic), and it contains fibres which indirectly, through the ciliary ganglion and short ciliary nerves, supply the non-striated sphincter pupillæ and ciliary muscle. The *fourth* nerve (patheticus) supplies the superior oblique alone, the *sixth* nerve (abducens) the external rectus alone.

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[1] Henschen, *Le Centre Corticale de la Vision.* Internat. Med. Congress, Paris, 1900.

Until comparatively recently, the motor nucleus for the pupil was generally considered to be located in the third nucleus, and in a special part of it near its anterior end (the so-called Edinger-Westphal nuclei, situated close to the middle line, one on each side, consisting of small nerve cells and embedded amongst the other larger cells of the oculomotor nucleus). And to explain the occurrence of loss of the light-reflex various theoretical lesions were assumed, sometimes in these Edinger-Westphal nuclei (Bernheimer [1]), sometimes in Meynert's fibres leading from the anterior corpora quadrigemina to the supposed pupillary centre in the third nucleus. But against the first of these theories, cases have been recorded of tumour of the mid-brain completely destroying the oculomotor nuclei and so producing ophthalmoplegia externa, and yet the pupillary reflex still remained (Biancone,[2] Jacobsen [3]). Moreover, total ophthalmoplegia (internal and external) has occurred without any affection of the Edinger-Westphal nuclei (Monakow [4]). Further, degeneration of Meynert's fibres has not been demonstrated, even in cases of tabes or general paralysis where loss of the pupillary light-reflex is one of the commonest clinical phenomena. Moreover, experimental and clinical evidence (Piltz,[5] Bach [6]) has shown not only that the *ciliary ganglion* is the peripheral motor nucleus controlling the sphincter pupillæ, but Marina,[7] in a series of twenty-eight cases of tabes and general paralysis exhibiting the Argyll-Robertson pupil, found this ganglion invariably degenerated, and in one of them where the Argyll-Robertson phenomenon was present in one eye, the ciliary ganglion was degenerated on that side alone, the ganglion of the other side being normal.

The Pupil.—We note the size of the pupil, both in a bright and in a dim light, and we observe whether the pupils are equal in diameter. Abnormal dilatation of the pupil (*mydriasis*) is often present in anæmia or neurasthenia, but it may occur, on one or both sides, in organic nervous disease. Mydriasis may be either paralytic, from paralysis of the sphincter pupillæ, as in disease of the third nerve or ciliary ganglion, or it may be irritative, as when due to stimulation of the dilator pupillæ. It is also seen when optic atrophy has caused blindness, and is then due to the absence of visual impressions. *Myosis* or abnormal contraction of the pupil occurs in pontine hæmorrhage, probably from irritation of inhibitory fibres leading from the brain to the ciliary ganglia.[1] It is also present in many cases of tabes, as well as in certain cases of disease of the cervical region of the cord (notably in syringomyelia) from interruption of the pupil-dilating fibres.[2] Myosis is also caused by iritis and by the irritation of foreign bodies in the cornea.

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[1] v. Graefe's *Archiv.*, 1897.

[2] *Rivista di Freniatria*, 1899.

[3] *Deutsche Med. Wochensch.*, 1900.

[4] *Gehirn-pathologie*, 4te Aufl. 1905, p. 1053.

[5] *Neurologisches Centralblatt*, 1903.

[6] *Zeitsch. für Angenheilkunde*, 1904.

[7] *Annali di Nevrologia*, 1901.

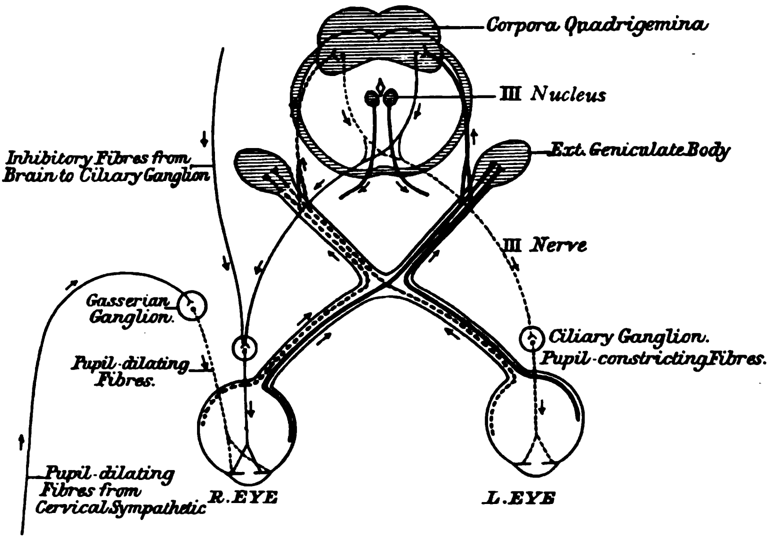

FIG. 39.—Diagram of the path of the pupil-reflex (modified from Bach).

Variations in the size of the pupil may also be the result of mydriatic drugs, either locally instilled (atropine, homatropine, cocaine) or taken internally (belladonna), whilst other drugs are myotics, either local (eserine, pilocarpine) or internal (opium, jaborandi).

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[1] Bach, *Zeitschrift für Augenheilkunde*, 1904, p. 105.

[2] See later, *Cervical Sympathetic*, p. 286.

The outline of the pupil should be carefully examined. Sometimes, instead of being circular, it is oval or irregularly polygonal. Such variations have an important diagnostic value. For if we exclude congenital malformation such as coloboma, operative procedures such as iridectomy, and disease such as iritis and synechiæ, then it may be taken as a general rule that irregularity of the pupils signifies either tabes, general paralysis, or old syphilis, the lesion being either in the short ciliary nerves or in the ciliary ganglion itself. Irregularity of the pupil can be produced experimentally by stimulation or division of the short ciliary nerves. Each eye must be tested separately, noting the effect on the pupil of shading and uncovering, first the same eye and then the opposite eye.

The *pupillary reflex to light* should always be observed. Normally the iris contracts when light falls on the retina, whether of the same eye (direct reflex) or of the opposite eye (consensual reflex). The light-reflex depends upon the integrity of a reflex arc, whose afferent limb is the retina and optic nerve, whose intermediate station is in the mid-brain and whose efferent limb passes through the third nerve and ciliary ganglion to the pupillary sphincter (Fig. 39).

Loss of reaction to light occurs therefore in optic atrophy, in paralysis of the third nerve, or in degeneration of the ciliary ganglion. Loss of the light-reflex with preservation of contraction during accommodation for near objects—the classic *Argyll-Robertson phenomenon*—occurs typically in tabes and in general paralysis of the insane. Marina has shown this to be associated with degeneration of the ciliary ganglion. It also occurs in blindness from optic atrophy. *Wernicke's hemiopic pupillary reaction* is absence of pupillary contraction in certain cases of hemianopia, when a ray of light is thrown on the blind side of the retina. It signifies a lesion of the visual path behind the chiasma, and below or at the corpora quadrigemina. In retro-quadrigeminal hemianopia, where the lesion is anywhere between the corpora quadrigemina and the visual cortex, the pupillary reaction is normal (Fig. 17).

The *reaction of the pupil to accommodation* is the contraction of the pupil which occurs when the patient converges the eyes to look at a near object. We test this by holding a finger close to the patient's face, first telling him to look at some distant object, and then suddenly to look at the finger. If he is blind, he can nevertheless converge by looking at his own finger. In paralysis of the third nerve there is total immobility of the corresponding pupil, both to light and on convergence. Loss of the contraction on accommodation with preservation of the light-reflex —a condition the converse of the Argyll-Robertson phenomenon —is not uncommon after diphtheria, and is often accompanied by other evidences of post-diphtheritic neuritis, such as paralysis of external ocular muscles or of the palate, loss of knee-jerks, &c. *Paradoxical pupillary reaction* is when the pupil dilates instead of contracting on accommodation. The *reaction of the pupil to painful stimulation* of the skin of the neck is important with regard to the cervical sympathetic (see later, p. 286).

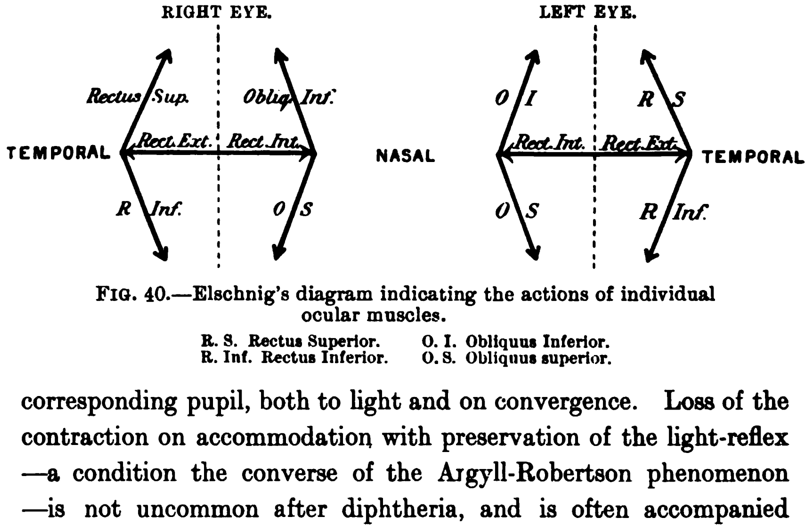

FIG. 40.—Elschnig's diagram indicating the actions of individual ocular muscles.

R. S. Rectus Superior. O. I. Obliquus Inferior.
R. Inf. Rectus Inferior. O. S. Obliquus superior.

Let us now consider the **paralyses of external ocular muscles**

To detect paralysis of the ocular muscles, having first examined the pupils, noting their size and any irregularity of outline, and having tested their reaction to light and on accommodation, we then ask the patient to follow our finger with his eye, looking alternately up, down, to the right and left, and converging. Meanwhile we observe whether there be any squint, deficient movement in any direction, diplopia, or nystagmus.

If an individual muscle is paralysed, there is diplopia, squint, and deficiency of movement of the affected eye towards the direction of traction of the affected muscle. Fig. 40 is Elschnig's diagram indicating the action of the individual muscles. A simple rule, worth remembering in all cases of ocular paralysis, is that the affected eye is displaced (by the unopposed antagonists) in a direction opposite to the direction of traction of the paralysed muscle, whilst the false image, seen by the affected eye, is displaced in the direction of traction of the paralysed muscle. Figs. 41 and 42 are Werner's well-known "memoria technica," showing the position of the false image in the various ocular paralyses. Fig. 41 shows the position of the false image in paralysis of any of the recti; fig. 42 in paralysis of the oblique muscles. For example, Fig. 41 shows that in diplopia from paralysis of the left inferior rectus, (1) the false image is on the right of the true (*i.e.* it is crossed); (2) the false image has its upper end inclined towards the true; (3) the false image is lower than the true; and (4) the diplopia occurs on downward movement of the eyes. To test diplopia we

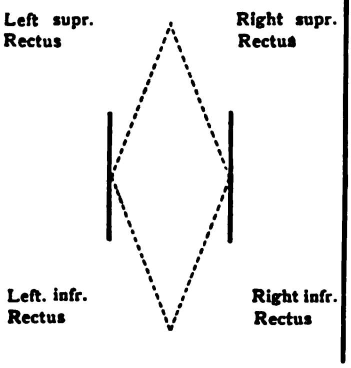

FIG. 41.

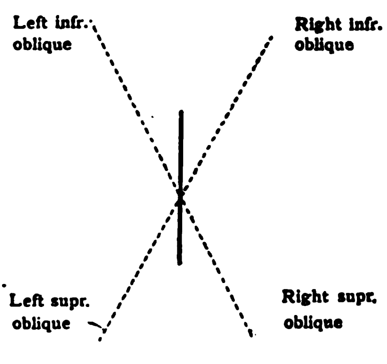

FIG. 42.

Figs. 41 and 42.—Werner's "artificial memory" for the double images in ocular paralyses (*Ophthalmic Review*, 1886). Fig. 41 shows the position of the images in paralysis of the recti muscles. Fig. 42 in paralysis of the oblique muscles. The dotted lines indicate "false" images, the thick black lines "true" images.

use a long lighted candle, at a distance of about three yards from the patient, holding it, first, exactly opposite the patient and moving it gradually from side to side and then from above downwards. One of the patient's eyes is covered with a red glass, to differentiate the two images, and the patient has to tell us the relative position of the red and of the white candle. It is necessary to ensure that the patient keeps his head fixed during the process of testing. The false image is that seen by the paralysed eye, the true image by the sound one.

Diplopia is a more delicate test than paralytic squint, for where there is slight paresis of an ocular muscle there may be no noticeable squint, and yet the diplopia may be quite appreciable to the patient. To detect a paralytic squint we direct the patient to follow our finger, moving it laterally from side to side, and then vertically up and down, and observe whether there is deficiency of movement of one or both eyes in any particular direction or directions.

Nystagmus is an involuntary rhythmic tremor of the eyeballs, generally bilateral and symmetrical. The movement consists of an oscillation usually laterally, from side to side, but it is sometimes vertical or even rotatory. In most cases nystagmus occurs only when the eyes are voluntarily moved to an extreme degree either laterally or vertically. But sometimes, especially in the rotatory variety, nystagmus occurs when the eyes are directed straight forward. In cases where an ocular muscle has been paralysed but is in process of recovery, if we make the patient look steadily in a direction which necessitates the active movement of the formerly paralysed muscle, slight oscillatory nystagmus may develope. The symptom occurs in various organic diseases, notably in disseminated sclerosis, Friedreich's ataxia and cerebellar disease. It is present also in certain patients who have become more or less blind (though in complete blindness the movement is more often a slow rolling of the eyes), also in albinism, and a well-recognised form is miner's nystagmus, due to persistent ocular strain in a dim light. In cases where an ocular muscle has been paralysed but is recovering, if we direct the patient to look steadily in a direction which necessitates the active innervation of the previously paralysed muscle, slight oscillatory nystagmus may develope, analogous to the tremulousness of the hand after carrying a heavy weight.

We are now in a position to recognise the symptoms of paralysis of any of the ocular nerves. In a case of complete **third nerve paralysis** (Figs. 43 and 44) there is ptosis or drooping of the upper lid, from paralysis of the levator palpebræ, with over-action of the frontalis on that side, so that the eyebrow stands higher than normal. In hysterical ptosis (Fig. 45), on the other hand, there is no over-action of the frontalis, nor is there in the ptosis of myasthenia gravis, where the frontalis is usually partially paralysed as well. In third nerve paralysis there is also external strabismus from unopposed action of the external rectus, and there is inability to move the eye upwards, directly downwards, or directly inwards, although a slight downward and inward movement can be executed by the superior oblique The pupil is dilated owing to paralysis of the sphincter iridis, and does not contract either to light or on attempted accommodation. Complete paralysis of the third nerve is less common than is a partial paralysis, affecting one or more muscles.

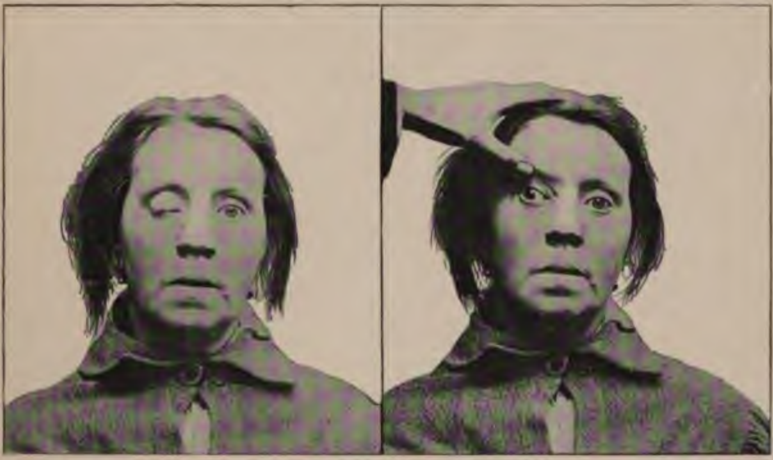

FIG. 43. FIG. 44.

Fig. 43.—Total paralysis of right third nerve from syphilitic disease.
Fig. 44.—The same patient, the right eyelid being passively lifted to show the external strabismus and dilatation of pupil on the paralysed side.

Paralysis of the Fourth Nerve causes impairment of the downward and inward wheel-rotation of the eye around its antero-posterior axis by the superior oblique. The deficiency of movement is difficult to see, and the paralysis is recognised mainly by the characteristic diplopia which occurs when the patient gazes in the direction in which the superior oblique ought to come into action, *i.e.* downwards and inwards. When the patient looks horizontally forwards or upwards there is no diplopia. But when he looks downwards and inwards, diplopia appears, the false image

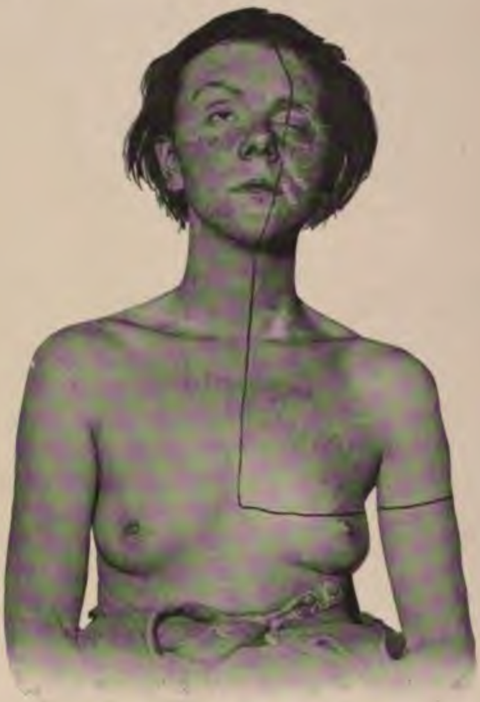

FIG. 45.—Case of left-sided hysterical ptosis in a woman of 25, showing absence of frontalis over-action on paralysed side. The area on the left side of the head, neck, trunk and shoulder, within the black line, was totally anæsthetic to all forms of stimuli. There was loss of smell and taste on the left side: contraction of the left visual field and diminution of hearing in the left ear. The figure also shows the presence of "dermographism." The patient's name having been traced on the chest with the head of a pin, a hard, cord-like pattern was produced, capable of being photographed.

standing lower than the true, and having its upper end tilted towards the other (Fig. 42). The false image also appears to the patient nearer to him than the true, the reason for which is obscure. Moreover the patient feels giddy, especially when he looks downwards, as in walking downstairs, and he habitually inclines his head forward and towards the sound side.

Paralysis of the Sixth Nerve is particularly easy to recognise. There is merely paralysis of the external rectus, with inability to turn the eye outwards beyond the mid-point, all other movements being normal (Figs. 46 and 46A), and there is diplopia on looking outwards. In old cases in which contraction of the non-paralysed internal rectus has supervened, an internal strabismus results.

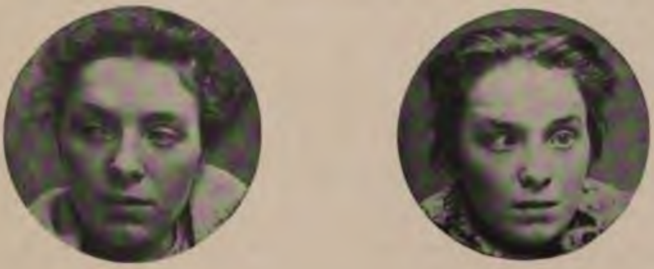

FIG. 46. FIG. 46A.

Case of paralysis of the left sixth nerve, of six weeks' duration, in a girl of 20, the subject of juvenile tabes.

Fig. 46 shows the normal movement of both eyes on looking to the right.
Fig. 46A shows attempted movement of eyes to the left. The left eye is arrested at the mid-position.

Sometimes an external ocular muscle is attacked by rheumatic myositis, which causes an ocular palsy of benign form. The muscle most frequently thus affected is the external rectus.

Ocular paralyses differ in type according as they are due to a supra-nuclear lesion (between the second frontal gyrus and the ocular nuclei), a nuclear lesion in the mid-brain, or an infra-nuclear lesion of the individual nerves such as we have just described.

Ocular paralysis from a supra-nuclear lesion never attacks a single ocular muscle or even a single eye. On the contrary, associated muscles of both eyes are affected. The common type of paralysis from a destructive lesion at or above the internal capsule is one in which the patient loses the power of turning both eyes towards the other side. Therefore the unopposed antagonists draw both eyes over towards the side of the lesion; this is called *conjugate deviation*. Curiously enough, conjugate deviation upwards and downwards does not occur from a paralytic lesion of the internal capsule, unless a bilateral lesion is present.

Skew deviation of the eyes occurs in certain lesions of the lateral lobe of the cerebellum or of its middle peduncle. Thus in a woman with a fatal hæmorrhage in the right half of the cerebellum and pons, the right eye was directed downwards and the left eye upwards.

A nuclear lesion of the third, fourth, or sixth nuclei in the floor of the Sylvian aqueduct may be partial or complete, and the type of ocular palsy which results is called **nuclear ophthalmoplegia.** In some cases the fibres for the ciliary ganglia or the ganglia themselves or short ciliary nerves to the internal ocular muscles (iris and ciliary muscle) are alone affected, and not the external muscles of the globe. The result is *ophthalmoplegia interna*, in which the pupils are dilated and immobile both to light and on convergence. This condition may be unilateral or bilateral, according as the ciliary ganglia or short ciliary nerves are affected on one or both sides. It often occurs as a transient result of post-diphtheritic neuritis. *Ophthalmoplegia externa* is a nuclear disease of the Sylvian aqueduct affecting numerous external ocular muscles, generally of both eyes and often symmetrically. A fairly common type is where the power of upward rotation of the eyes is lost, lateral movements being still possible. Ophthalmoplegia externa usually occurs alone, less commonly it is associated with the internal variety. When both varieties are combined we have *total ophthalmoplegia*, in which both eyes are fixed and motionless, the pupils being immobile, and the patient can look in any particular direction only by facing his head that way *en bloc*. Nuclear ophthalmoplegia, especially external ophthalmoplegia, may be associated with motor paralysis of the limbs if the lesion extends ventrally and implicates one or other pyramidal tract, or it may be associated with involuntary tremors if the lesion affects the red nucleus or rubrospinal tract (Fig. 12, p. 18).

Sometimes it is possible to differentiate between a nuclear and an infra-nuclear ocular lesion. In the case of *paralysis of the sixth nucleus* in the pons, there is not merely weakness of the external rectus of the same side, as in paralysis of the sixth nerve trunk, but in addition the internal rectus of the opposite eye is paralysed, so that conjugate movement of both eyes towards the affected side is impaired. The weakness of the contralateral internal rectus is only in connection with its associated movement with the external rectus of the homolateral eye. This is proved by the fact that, in paralysis limited to the sixth nucleus, both internal recti can still act normally during convergence. Again, since the facial nerve loops around the sixth nucleus within the pons, a lesion of the sixth nucleus is not infrequently accompanied by facial paralysis on the same side.

With regard to the diagnosis between nuclear and infra-nuclear paralysis of the third nerve, if in a doubtful case the orbicularis oculi is found to be affected together with the external ocular muscles, then the lesion is nuclear, since the orbicularis is innervated by fibres which, starting from the lower end of the oculomotor nucleus, reach the muscle, not through the third nerve, but through the facial nerve.

Sometimes transient ocular palsy affects the third nerve in whole or in part, recurring in the same eye without apparent cause at intervals of weeks or months, and clearing up completely between the attacks. This condition, known as Charcot's *migraine ophthalmoplegique*, is generally associated with headache, most intense in the eye and forehead of the affected side, and with vomiting. Its pathology is obscure; probably some cases are due to an inflammatory affection of the meninges at the point where the third nerve pierces them to enter the sphenoidal fissure. This is all the more probable inasmuch as the first division of the fifth nerve, which traverses the sphenoidal fissure, is often simultaneously affected with the result that there is blunting of sensation in its area of distribution.

Now and then we meet with ***congenital ptosis***, in which there is paralysis of the superior rectus and levator palpebræ superioris of one eye. In such cases, although the patient cannot raise his upper lid voluntarily, yet, curiously enough, the lid is raised when certain jaw-movements are made, particularly when the patient throws into action the external pterygoid muscle of the same side in depressing the lower jaw.

The **Fifth or Trigeminal Nerve** has a most extensive distribution, the main points of which may here be recapitulated. The nerve consists of two distinct parts, sensory and motor. The sensory root, the one on which is the Gasserian ganglion, divides below the ganglion into three divisions, of which the first two are entirely sensory. The motor root courses beneath the Gasserian ganglion, and then joins the third division, which thus becomes a mixed nerve.

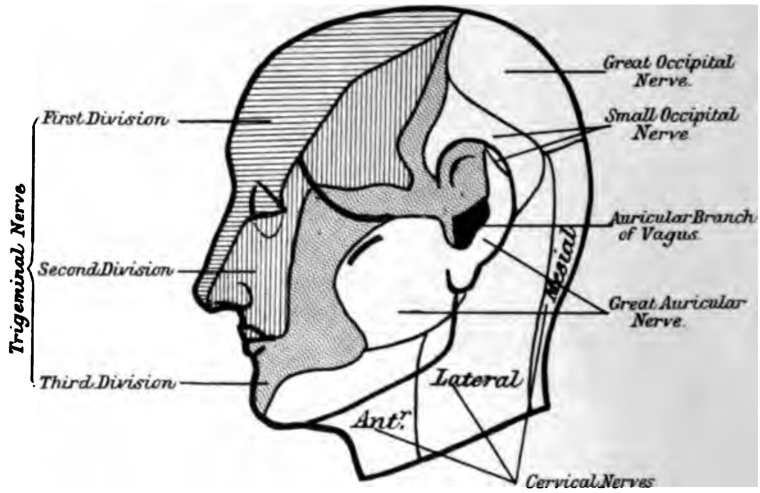

FIG. 47.—Cutaneous supply of head (Frohse).

The *first or ophthalmic division* passes through the sphenoidal fissure into the orbit and supplies the eyeball and lachrymal gland, the conjunctiva (except that of the lower lid), the skin of the forehead and scalp up to the vertex (Fig. 47), the mesial part of the skin of the nose, and the mucous membrane of the upper part of the nasal cavity. It also contains efferent pupil-dilating fibres derived from the cervical sympathetic, joining it at the Gasserian ganglion, and going to the iris (Fig. 39).

The *second or superior maxillary division* passes through the foramen rotundum across the spheno-maxillary fossa to the infra-orbital canal. In the spheno-maxillary fossa it is connected with Meckel's ganglion, which gives off amongst other branches the Vidian nerve. This latter runs backwards to join the facial nerve, the posterior end of the Vidian being named the great superficial petrosal (Fig. 48). The superior maxillary division supplies the skin of the upper lip, the side of the nose and adjacent part of the cheek, the lower eyelid and part of the temple. It also supplies the conjunctiva of the lower lid, the upper teeth, the mucous membrane of the upper lip, the upper part of the cheek, upper jaw, uvula, tonsil, naso-pharynx, middle ear and lower part of nasal cavity. It also contains some taste fibres to which we shall refer presently.

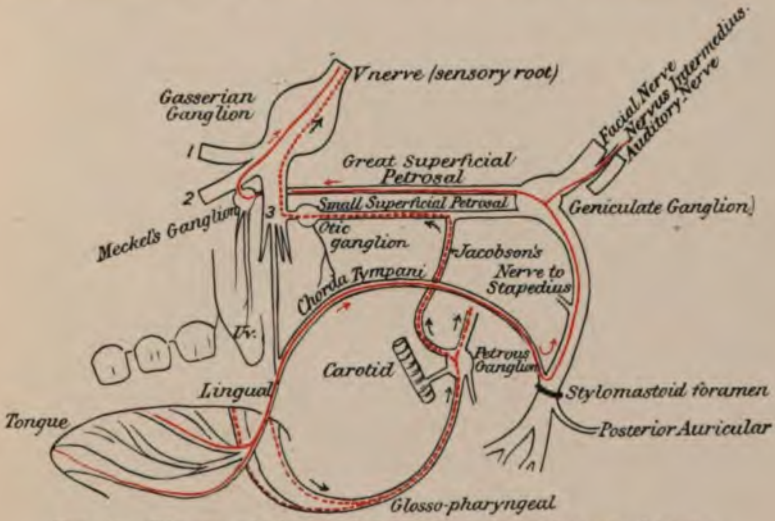

FIG. 48.—Diagram of trigeminal, facial, and glosso-pharyngeal nerves, showing course of taste-fibres (modified from Leube).

The *third or inferior maxillary division* is a mixed nerve. It emerges through the foramen ovale. The motor fibres supply the masseter, temporal, and both pterygoid muscles, also the tensor tympani, mylo-hyoid and anterior belly of digastric. The sensory fibres supply the skin of the posterior part of the temple and adjacent part of the pinna, the anterior and upper wall of the external auditory meatus, as far as and including the anterior part of the drum, part of the cheek, the lower lip and chin, also the lower teeth and gums, the tongue (as far back as the circumvallate papillæ), floor of mouth, inner surface of cheek, and salivary glands. This division also contains some taste fibres at its origin from the Gasserian ganglion.

The **Course of the Taste Fibres** is a complicated one (Fig. 48). Those for the anterior two-thirds of the tongue are contained in the lingual nerve—a branch of the third division. But they do not run straight up from the lingual into the fifth nerve. They leave the lingual, course along the chorda tympani, and reach the facial within the Fallopian aqueduct. They run in the facial as far as the geniculate ganglion, where some pass off along the great superficial petrosal to Meckel's ganglion, ultimately rejoining the fifth nerve through its second division. Other taste-fibres leave the facial to enter the nervus intermedius, passing thence to the glosso-pharyngeal nucleus.

The taste fibres for the posterior third of the tongue and the palate, which are supplied through the glosso-pharyngeal nerve, probably do not enter the brain entirely through the glosso-pharyngeal. Some of them join the fifth nerve, since division of the fifth nerve by the operation of removal of the Gasserian ganglion causes impairment of taste, not only in the anterior two-thirds but in the posterior third of the tongue. Taste is not entirely abolished, as was formerly thought, for in several cases of my own I have found that the patient, though unable to feel the contact of food or other objects on one side of the tongue, still retained some sense of taste. The route of the fibres from the glosso-pharyngeal to the fifth is probably through the tympanic plexus (Jacobson's nerve) to the small superficial petrosal, and thence through the otic ganglion to the third division of the fifth.

To examine the sense of taste we direct the patient to protrude the tongue, and we rub on it various substances such as sugar, salt, quinine, and citric acid, preferably in white powders, which the patient cannot distinguish at sight one from the other. The patient must keep his tongue protruded throughout each test, and as soon as he feels a taste he should make a sign and also determine in his mind what the taste is, before taking the tongue in. Otherwise if he be allowed to pull the tongue in while waiting for the sensation to arrive, fallacies may occur owing to movements of the tongue and the flow of saliva carrying the substance to other parts. To map out exactly an area of loss of taste (*ageusia*) the most accurate method is to use a weak galvanic current with a wire electrode, which produces a coppery or metallic taste.

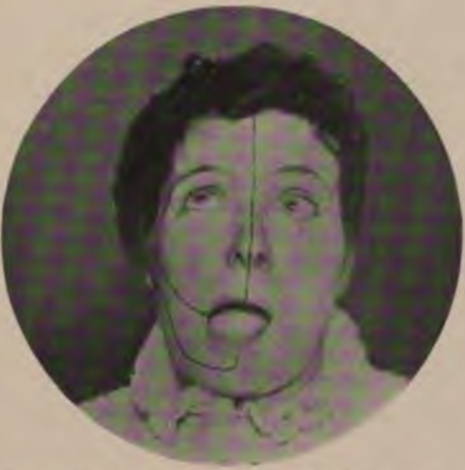

FIG. 49.—Paralysis of Left third and of Right fifth nerve. The patient is looking upwards and opening the jaw. Showing dilatation of pupil on left side with deficient upward movement of left eye. Also showing the deviation of the lower jaw towards the right side. The black line indicates area of anæsthesia on the right side of the face.

When the fifth nerve is totally paralysed there is anæsthesia of the corresponding half of the face and scalp, not extending as far as the angle of the jaw, this part being supplied by the cervical plexus (Fig. 47). The cornea and conjunctiva on the affected side are anæsthetic, and also the mucous membrane of the corresponding side of the nose, mouth, part of the soft palate and tongue, as far back as the circumvallate papillæ which, with the area behind, are innervated by the glosso-pharyngeal. This defect extends exactly to the middle line, and therefore the patient when drinking feels as if the cup were broken. Food tends to collect within the anæsthetic cheek, the buccinator muscle being anæsthetic, though its motor power is unimpaired. Taste is generally impaired in the anterior two-thirds of the tongue, and to a less extent at the back, but does not remain totally lost. The trigeminal nerve has also sensory fibres for the facial muscles. Hence there is a degree of awkwardness and apparent weakness of the face—a pseudo-facial palsy, due to loss of the sense of active muscular contraction. All the muscles supplied by the motor root undergo atrophic paralysis and develope the electrical reactions of degeneration. There is hollowing of the temporal fossa above the zygoma, and wasting of the masseter below it, so that the zygoma becomes abnormally prominent. When the patient clenches his teeth, neither the temporal nor the masseter can be felt to harden as on the normal side, and when he opens his mouth the mandible swings over towards the paralysed side (Fig. 49). This is owing to paralysis of the external pterygoid, which fails to draw the condyle forwards on the affected side. The deflected mandible carries with it the tongue, but there is no real deviation of the tongue, when measured from the middle line of the lower incisors. Paralysis of the tensor tympani is stated to cause a difficulty in hearing notes of low pitch, but this is not easy to determine. Secretion of tears on the paralysed side is diminished, as is also the secretion of nasal mucus and of saliva. Consequently these mucous membranes become abnormally dry, and may show secondary trophic changes. Thus stimulation of the nasal mucous membrane by snuff no longer causes sneezing. Smell at first is unimpaired, but later, from dryness and secondary trophic changes in the Schneiderian membrane, there is complete anosmia in the affected nostril. The corneal and the lachrymal reflex are lost, also the palatal reflex, and the tongue on the paralysed side becomes excessively furred, probably because on the anæsthetic side there is deficient friction by food. The teeth on the paralysed side are anæsthetic and tend to drop out; this has been ascribed to a trophic change, but more probably it is mainly traumatic, the patient biting clumsily with his anæsthetic teeth. It used to be stated that neuro-paralytic ophthalmia occurred in total trigeminal palsy, owing to trophic changes. But this is not invariably so; and when it does occur, it is probably because some irritant gains access to the anæsthetic eye, where, moreover, there is a deficiency of lachrymal secretion.

There is another disease which occurs in the territory of the fifth nerve, viz., **progressive facial hemiatrophy.** This disease, which commences in early life—usually before puberty, and more often in females than in males—shows itself first in the skin of the face, either near the orbit or over the upper or lower jaw, gradually spreading over the whole face on one side. The skin becomes thinned from atrophy of its papillary layer, the subcutaneous fat disappears, and thus the affected side of the face becomes wrinkled and furrowed, in marked contrast with the healthy side. Later the subjacent muscles, cartilages, and bones become atrophic, but without motor paralysis or reaction of degeneration. The corresponding side of the tongue (Figs. 51 and 130), and occasionally

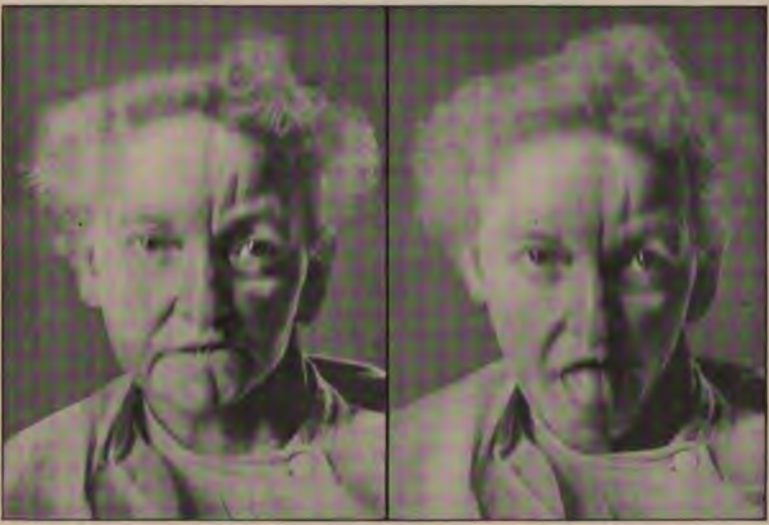

FIG. 50. FIG. 51.

From a case of left-sided facial hemiatrophy. Showing atrophy of corresponding half of tongue.

that of the soft palate, also becomes wasted. But the hemi-atrophied tongue, when protruded, comes out straight, unlike that of a case of atrophy from hypoglossal palsy (Fig. 65, p. 141). The hair on the affected side of the face may fall out or become white, and the sebaceous glands atrophy. The scalp is rarely affected. There is no anæsthesia. The area of this disease corresponds accurately with that of the distribution of the fifth nerve, and in certain cases pathological changes have been found either in the nerve itself or in its nucleus of origin. Thus Mendel found signs of neuritis in the nerve, together with changes in the spinal root of the fifth within the medulla, while more recently Loebl and Wiesel[1] found an interstitial neuritis of the Gasserian ganglion and of the parts distal to it.

[1] *Deutsche Zeitschrift für Nervenheilkunde*, 1904, Bd. 27, s. 355.

LECTURE X

CRANIAL NERVES (*continued*)

Of all the peripheral nerves in the body, cranial or spinal, the **Seventh or Facial nerve** is by far the most frequently paralysed. Hence the importance of knowing its anatomical course and distribution.

Arising from a nucleus in the lower part of the pons, but deriving some of its fibres, namely those for the orbicularis oculi, from the nucleus of the third nerve, and others, namely those for the orbicularis oris, from the hypoglossal nucleus, the facial nerve pursues a tortuous course. Firstly, within the substance of the pons, it forms a loop which hooks round the nucleus of the sixth nerve. Then, leaving the ventral surface of the medulla, it enters the internal auditory meatus, and passes along a winding bony canal in the temporal bone—the aqueduct of Fallopius. In the upper part of this canal it has a swelling, the geniculate ganglion, at which point it is joined by the portio intermedia of Wrisberg, by the great superficial petrosal nerve from Meckel's ganglion, and by the small superficial petrosal from the otic ganglion (see Fig. 48). Within the aqueduct it gives off a branch to the stapedius, and lower down the chorda tympani leaves it to join the lingual nerve. It then emerges from the skull through the stylo-mastoid foramen, gives off a posterior auricular branch to the muscles of the pinna and to the occipital belly of the occipito-frontalis. The main trunk then divides into its terminal branches supplying all the muscles of the face (except the levator palpebræ superioris) from the frontalis above to the platysma below. It also supplies the stylo-hyoid and posterior belly of the digastric.

Although the facial nerve is mainly motor, it also contains certain secretory fibres, and the chorda tympani taste-fibres accompany the nerve in part of its course. Thus lesions at different levels can be distinguished one from the other.

1. If the facial nerve is affected *after its exit from the stylo-mastoid foramen*, *e.g.* by cold, or by injuries or tumours in that region, the result (**Bell's Paralysis**) is complete palsy of that side of the face, which is therefore asymmetrical at rest, and the asymmetry is exaggerated on voluntary movement. The patient has neither emotional nor voluntary movement of the affected side (Figs. 52 and 53).

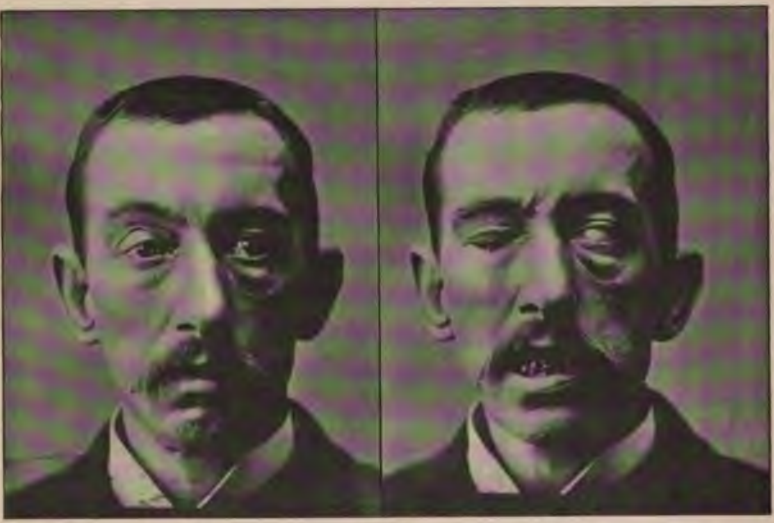

FIG. 52. FIG. 53.

Case of left-sided facial palsy. Fig. 52 at rest. Fig. 53 on attempt to close eyes and retract angles of mouth.

The furrows of the forehead are wiped out, and the patient cannot wrinkle the brow nor frown on that side. The eye is more widely open on the affected side and cannot be shut. The tears run down the cheek instead of into the lachrymal duct, and may produce excoriation of the skin or eczema. When the patient tries to shut the eye he merely rolls the eyeball upwards and outwards, or upwards and inwards, sometimes with a zig-zag movement, until the cornea passes under cover of the upper lid.

An additional sign pointed out by Dutemps and Cestan[1] is as follows :—When the patient looks down and then attempts to shut both eyes slowly, the upper lid on the paralysed side is seen to move up a little, owing to contraction of the levator palpebræ, which normally acts "synergically" with the orbicularis but is now no longer antagonised by it.

This inability to close the eye allows the entrance of foreign bodies, and consequently conduces to conjunctivitis. The conjunctival reflex is abolished, and the regular involuntary blinking of health no longer occurs on the paralysed side. The eye brims over with tears, so that vision on the affected side is rendered less acute. Curiously enough, though the eye cannot be shut during waking hours, during sleep it often closes almost completely, probably from relaxation of the levator palpebræ.

The tip of the nose is drawn somewhat towards the sound side, the naso-labial fold on the affected side is flattened out, the ala nasi sinks in and shows no active movement, voluntary or respiratory, though it may flap loosely during forcible nasal breathing. The mouth is drawn towards the sound side, but on the affected side its angle droops and saliva dribbles from it. When the patient smiles or shows the upper teeth, the healthy side moves alone ; he cannot whistle, and the articulation of labial consonants is impaired. During mastication food accumulates between the teeth and the paralysed cheek. The patient often bites his cheek or lower lip, and during forcible blowing expiration the paralysed cheek flaps loosely. Voluntary movement of the integument by the platysma, as in forcible depression of the chin against resistance, is abolished on the affected side. In those patients who were previously able to move the ear voluntarily, that power is also lost. All the paralysed muscles gradually develope the electrical reactions of degeneration. The affected side of the face generally sweats less than the healthy side.

2. If the lesion be *within the Fallopian aqueduct* below the geniculate ganglion, it produces all the above symptoms, and, in addition, from implication of the chorda tympani, there is loss of taste, (and sometimes slightly of common sensation,) in the

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[1] *Journal de Neurologie*, 1904, p. 48.

anterior two-thirds of the tongue on the affected side, and also occasionally abnormal subjective sensations of taste, and sometimes diminution or excess of submaxillary and sublingual saliva. The

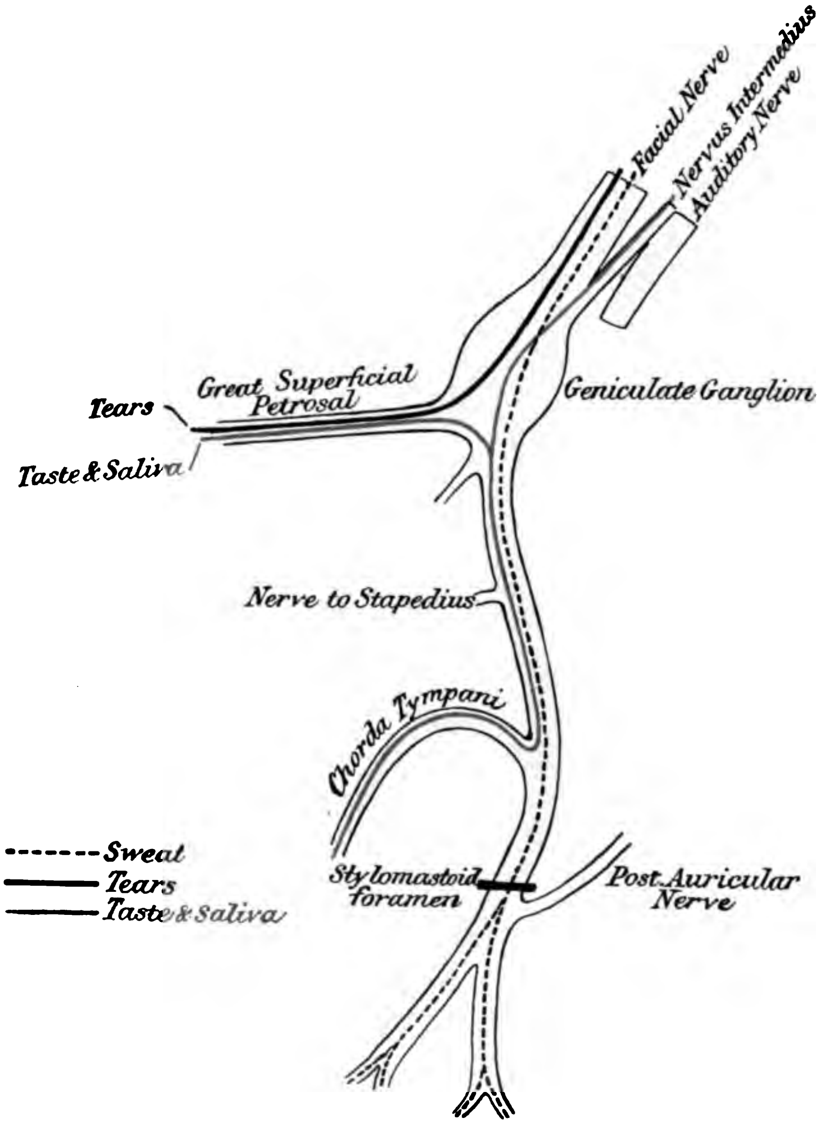

FIG. 54.—Diagram of Facial Nerve, showing course of secretory and of taste fibres.

deficiency of taste and of saliva may cause this part of the tongue to be abnormally furred up to the middle line. If there be paralysis of the nerve to the stapedius, there is hyperacousis or painful sensitiveness to musical notes of low pitch (presuming that the auditory apparatus is not affected), and the patient can no longer produce the subjective noise in the ear, which we normally hear on attempting very forcibly to innervate the facial muscles, especially the orbicularis palpebrarum.

3. If the nerve be affected *between its emergence from the pons and the geniculate ganglion*, it produces the same symptoms as in Bell's paralysis, but without affection of taste in the front of the tongue. And since disease in this region almost invariably implicates the auditory nerve, there is usually deafness also. If the auditory nerve chances to escape, hyperacousis will occur from stapedius paralysis. Many cases have deficiency of tears on the affected side. Most cases of basal intra-cranial disease present general symptoms also, such as headache, giddiness and vomiting.

4. If the lesion of the nerve be *within the substance of the pons*, facial palsy results as in Bell's paralysis, but taste and hearing are unaffected. There is, however, usually an accompanying paralysis of the sixth nerve or its nucleus, since the facial nerve hooks round the sixth nucleus within the pons.

Paralysis of the soft palate used to be included in the symptoms of a lesion of the facial nerve at or above the geniculate ganglion. But the weight of evidence goes to prove that the facial has no share in the innervation of the palate. Chvostek,[1] in 1883, published a case of sarcoma of the facial nerve in which paralysis of the palate had been observed, but that was before the days of the Marchi method, and it is impossible to be sure that the lower roots of the vagus were undegenerated. In his case there was also a cancer in the tongue.

Slight cases of facial palsy, whether due to cold, middle-ear affection, compression, or other causes, may recover completely in one or two weeks. More severe cases last from two to eight months before recovery begins. Or the palsy may remain permanent. In severe cases, where improvement does not begin for three months or more, a spastic or contractured condition usually comes on as voluntary power reappears. The mouth becomes drawn back again towards the paralysed side, the palpebral fissure instead of being wider, is narrower than on the healthy side, and the naso-labial and other furrows not only reappear, but become exaggerated. The result is that, when a rest, the healthy side may seem the weaker of the two, though when voluntary movement takes place it is easy to see which is the affected side. Together with this contracture there is always in the spastic muscles a tendency to over-action, imperfect recovery being associated with imperfect control. One variety of facial hemi-spasm results (see p. 77). Thus, on closing the eye on the affected side, the angle of the mouth becomes drawn outwards; or again, on showing the upper teeth, the eye becomes closed. Sudden flickers of involuntary fibrillary tremors may sometimes be seen on the affected side.

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[1] *Wiener Medizinische Presse*, 1883, p. 34.

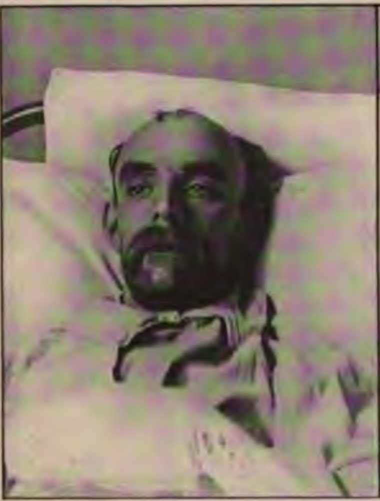

FIG. 55.

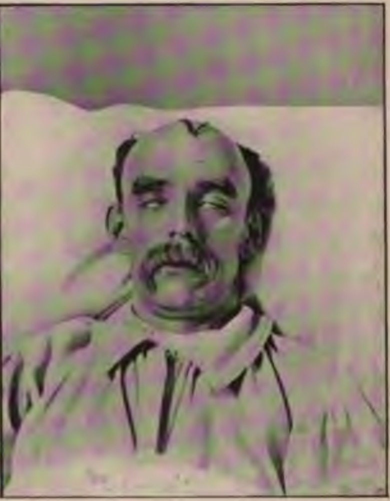

FIG. 56.

Fig. 55.—Bilateral facial palsy, alcoholic in origin, associated with peripheral neuritis of upper and lower limbs.
Fig. 56.—Maximum voluntary movement of face, on attempt to close the eyes and to retract angles of mouth.

Secondary contracture occurs only in cases of incomplete recovery. During the stage of total paralysis, when no impulses are reaching the muscles, they are quite flaccid. The development of contracture indicates that muscular regeneration has been imperfect.

Bilateral facial palsy is rare. It may be due either to intra- or extra-cranial causes, the commonest intra-cranial cause being gummatous basal meningitis. Of the extra-cranial causes, the most important are double otitis media, cold, and post-diphtheritic paralysis. Alcoholic paralysis rarely attacks the facial nerve, but when it does so, the affection is bilateral, as in the case of the man shown in Figs. 55 and 56, who also had typical alcoholic neuritis of the limbs.

In bilateral facial palsy there is no asymmetry of the face, but it hangs like a fixed expressionless mask, incapable of evincing the slightest emotion.

Bilateral facial weakness also occurs in the "facio-scapulo-humeral" type of myopathy to which we shall refer in a later lecture.

The **Eighth or Auditory Nerve** comprises *cochlear fibres* for the function of hearing, and *vestibular fibres* which supply the semicircular canals and constitute the most important nerve of equilibration, which informs us of the position of our head in space. Affections of the cochlear fibres produce auditory phenomena, while disease of the vestibular fibres causes one form of vertigo, though vertigo and other auditory symptoms often result not only from disease of the labyrinth or vestibular fibres, but from affections of the middle or even of the outer ear.

The chief symptoms referable to the auditory nerve are deafness, tinnitus, and vertigo.

In a patient who is apparently deaf, we should always, before proceeding to test the hearing, examine the external auditory meatus, to make sure that it is not blocked by wax. We then test *aerial conduction* by the ticking of a watch, the patient's eyes being shut and one ear closed while the other is being tested. Holding the watch at some distance from the ear, we slowly bring it nearer until the patient can just detect the tick. If there is deafness, we have to determine whether this is due to middle-ear disease or to affection of the labyrinth or auditory nerve. The *tuning-fork tests* help us here. Normally a vibrating tuning-fork placed on the centre of the forehead is heard equally in both ears (Weber's test). If placed on the mastoid process, we wait till it is no longer conducted through the bone, and find normally that it is still audible when held close to the external meatus (Rinne's test). If the middle ear be diseased, or if the outer ear be blocked up, there is loss of aerial conduction, but bone-conduction is still preserved. The tuning-fork on the forehead is then heard louder on the affected side ("positive-Weber,") and Rinne's test is negative, *i.e.* the tuning-fork is no longer heard aerially after fading away on bone-conduction. But if the deafness be due to affection of the internal ear or of the auditory nerve—so-called "nerve-deafness," a tuning-fork on the vertex is not heard on the affected side ("negative-Weber"), whilst as a rule there is "positive-Rinne," though not always. In nerve-deafness the hearing is generally better in the midst of a noise (*e.g.* in an omnibus or tram) than in a quiet place, whereas in bilateral deafness from chronic middle-ear disease, the reverse is the case. Additional localising evidence may also be obtained from the other concomitant symptoms. Thus gross disease of the auditory nerve within the skull, *e.g.* in a case of lateral extra-cerebellar tumour, is often accompanied by facial paralysis, though this conjunction is of value only when middle-ear disease can be excluded. On the other hand, disease of the labyrinth is often associated with tinnitus or vertigo. Disease of the auditory nuclei in the pons may be associated with paralysis of the opposite arm and leg.

Tinnitus, or ringing in the ears, is a subjective symptom. It signifies irritation of some part of the auditory apparatus. The term does not include elaborate auditory hallucinations of cortical origin, such as distinct melodies or voices uttering intelligible words. The nature of the sound in tinnitus varies in different cases: for example, it may be buzzing, beating, or whistling. Broadly speaking, we recognise two main kinds of tinnitus—the pulsating and the continuous. *Pulsating* sounds, synchronous with the pulse, occur in some intra-cranial aneurisms, but are also not infrequent in simple neurasthenia in the "silent watches of the night." Curious "clicking" sounds in the ear may result from clonic spasm of the tensor tympani muscle. *Continuous* sounds may be of high or low pitch. We should always notice whether they are increased or diminished by the recumbent posture. Low-pitched continuous tinnitus may be the result of venous hyperæmia, in which case it is aggravated by recumbency, or of simple anæmia, which is relieved by lying down. Nitrite of amyl aggravates tinnitus when due to hyperæmia and relieves it when due to anæmia. High-pitched continuous tinnitus is generally due to labyrinthine stimulation, either from outer or middle ear affection (perhaps merely wax or water in the external meatus, or an indrawn tympanic membrane), or from actual labyrinthine disease. It is also caused by certain drugs, notably by quinine and salicylates. Pulsating tinnitus due to arterial congestion can often be arrested temporarily by compression of the vertebral artery supplying the labyrinth, or of the carotid supplying the external or middle ear.

Vertigo, or giddiness, is the peculiar disagreeable sensation which results if our sense of secure equilibration is disturbed. The process of equilibration is a muscular act, where all the muscles are innervated, of course, by the cerebral cortex, this latter being again largely influenced by the cerebellum. The cerebellum is a co-ordinating centre for equilibration. It receives afferent impulses from various sources, of which the semicircular canals of the inner ear are by far the most important, the others coming from the skin of those parts on which the body happens to be resting, from the muscles and joints concerned in maintaining our balance, and from the muscles of the head and eyes concerned in looking towards surrounding objects. Each half of the cerebellum exercises a co-ordinating influence, through the corresponding superior cerebellar peduncle, upon the contra-lateral cerebral cortex.

Giddiness is often accompanied by a feeling of movement either in the patient himself (subjective vertigo) or in external objects (objective vertigo). Severe giddiness usually produces the motor phenomenon of reeling or staggering.

Vertigo may result from affection either of the higher cerebral centres or of the co-ordinating cerebellar centres, or from affection of any of the afferent paths to which we have already referred. Severe vertigo is often accompanied by nausea and vomiting, as in sea-sickness.

Vertigo may occur in healthy people. Thus, for example, a galvanic current passed transversely through the head produces a variety of giddiness due to cerebellar stimulation. In this the patient tends to fall towards the side of the positive pole, and his head and eyes are also rotated in that direction, except at the moment of stopping the current, when he tends to fall towards the side of the negative pole. Rapid rotation of the body round its own axis, as in waltzing, or rapid changes in our position in space, as in swinging, produce giddiness which is probably due to variations in the pressure of the endolymph within the semicircular canals. Some people feel giddy when stepping unexpectedly from a firm surface on to a piece of boggy turf, or, as in a famous Edinburgh street, on to a piece of indiarubber pavement, this variety of vertigo being due to deficient sense of resistance conveyed from the skin of the soles and from the muscles and joints of the lower limbs. The giddiness produced by standing near the edge of a cliff or of a high tower is most probably due to loss of muscular impression from the ocular muscles. Ordinarily we have surrounding objects at or above our own level with which to compare our position in space, and if such objects are absent vertigo may result.

Vertigo is also associated with various pathological conditions. Among the intra-cranial causes we may mention blows on the head, and sudden *cerebral anæmia or hyperæmia.* A distinguished member of the medical profession who was the subject of aortic regurgitation used to have attacks of intense vertigo if he took a saline aperient. Probably in his case the withdrawal of a considerable amount of fluid from the circulation rendered the brain anæmic—hence the vertigo. It was always relieved by the recumbent posture, whilst cardiac tonics and the avoidance of hydragogue cathartics prevented its recurrence. Vertigo from cerebral hyperæmia is very common in women about the menopause. It is still more marked in many cases of cerebral hæmorrhage or thrombosis, of which it may be a premonitory signal. Giddiness in old people with atheromatous arteries, if it be associated with headache, and especially if there be no sign of labyrinthine disease, should always be regarded with caution. Intra-cranial tumours may cause giddiness by raising the general pressure within the skull, and cerebellar tumours are especially associated with vertigo, even apart from increased intra-cranial pressure. *Intra-cerebellar tumours* produce a vertigo in which the subjective sense of rotation of the body is in the same direction as that of the apparent movement of surrounding objects, *i.e.* away from the side of the lesion. In *extra-cerebellar tumours*, whilst external objects appear to move away from the side of the lesion, the sense of subjective rotation is reversed, *i.e.* towards the side of the lesion.

A characteristic form of vertigo has also been described by Bruns,[1] and confirmed by various other observers.[2] It is produced by the presence of a *cysticercus in the fourth ventricle.* Sometimes the worm is anchored to the ependyma, sometimes it is swimming free. The patient, who otherwise shows no sign of intra-cranial organic disease, has paroxysms of violent vertigo, chiefly on sudden movement of the head, either active or passive, causing a temporary shifting of the position of the worm. He also has attacks of occipito-frontal headache with vomiting; his gait is tottering and unsteady, and glycosuria is not uncommonly present. There may be intervals during which he is apparently well. Death usually occurs suddenly from respiratory paralysis.

Vertigo is associated with certain degenerative diseases, notably with disseminated sclerosis. Vertigo is frequently the "aura" of an epileptic fit, or may accompany the headache of an attack of migraine. A hereditary family form of giddiness has also been described.

[1] *Centralblatt für Neurologie*, 1902, p. 565.

[2] Osterwald, *Neurologisches Centralblatt*, 1906, p. 265.

Toxic vertigo from alcohol or tobacco is a familiar type, and to the toxic class we may also refer cases produced by gastric disorder, by constipation, and by some cases of intestinal parasites, though in the last instance a reflex element may also be present. Giddiness is often present in neurasthenic and hysterical patients, in whom it may be elicited by the slightest exciting cause, for example by rectal examination.

Ocular vertigo occurs in cases of paralysis of any of the external ocular muscles, and is associated with diplopia. The visual field being erroneously projected, the patient judges wrongly as to the relation of his body to what he sees. "Objects appear to be in certain positions where the patient's feet, as a matter of fact, fail to find them" (Hughlings Jackson). The giddiness in such cases is not due directly to the diplopia, for it persists when the sound eye is covered. The condition can be imitated in health by closing one eye and displacing the other eye inwards by pressure with the finger, when if the subject tries to walk along a straight line his gait becomes very unsteady.

But the vast majority of cases of vertigo are associated with some disorder of the ear. It may result from wax, or from foreign bodies in the meatus, or it may supervene during ear-syringing, especially if there is a perforation of the drum. Or the pathological cause may be in the middle ear, as in otitis media, obstruction of the Eustachian tube, or merely from sneezing or blowing the nose, also in spasm of the tensor tympani muscle.

Lastly, there is **Ménière's disease,** or labyrinthine vertigo. This has three main classes of symptoms; firstly, giddiness and reeling, due to affection of the semicircular canals; secondly, deafness and tinnitus, due to affection of the auditory fibres; and thirdly, associated bulbar phenomena such as cardiac failure, cold clammy sweat, nausea, and vomiting, due to affection of adjacent medullary centres.

The vertigo of Ménière's disease is paroxysmal, and comes on with such suddenness that the patient may fall to the ground as if struck down by an unseen hand. In other cases he reels, but has time to clutch at some neighbouring object to prevent himself from falling. The giddiness lasts sometimes for hours;

slighter attacks may pass off in a few minutes. It is increased by movement, and the slightest attempt to raise the head may induce vomiting. The direction in which the patient falls is usually forwards or towards one side, and commonly away from the side of the affected ear. Not infrequently nystagmoid jerks of the eyes occur during the attack, and double vision has also been observed. The vertigo is frequently accompanied or followed by headache, nausea and vomiting, lasting sometimes for hours. Together with these there are characteristic auditory phenomena, generally a sudden loud noise, usually unilateral. And there is deafness, more or less complete, on the same side as the tinnitus, with diminution or loss of bone-conduction. A certain degree of deafness remains between the attacks, but is rarely absolute.

Such symptoms, occurring with apoplectiform suddenness, constitute the typical picture of Ménière's disease. But frequently the paroxysms are much slighter, and unassociated with nausea or vomiting, so that the patient may simply have sudden transient giddiness. But the attacks tend to recur; rarely does a patient escape with a single attack. The intervals between them vary; they may gradually decrease in frequency, or may progressively increase until after successive attacks the deafness becomes absolute. The vertigo then usually ceases.

Ménière's disease is distinguished from ordinary epilepsy by the coexistence of vertigo, tinnitus, and deafness. Loss of consciousness, which is the rule in epilepsy, is rare in labyrinthine vertigo. From cerebral hæmorrhage or thrombosis it is distinguished by the presence of auditory phenomena, and by the absence of signs of a focal brain lesion. The pathology of Ménière's syndrome is obscure. Ménière himself described a hæmorrhagic effusion in the inner ear. But as Arthur Cheatle has luminously suggested, the phenomena are in many respects closely analogous to those of glaucoma, and may possibly be due to a sudden rise of tension in the endolymph or perilymph, whether produced by hæmorrhage or by other causes.

[1] *Archives of Otology*, vol. xxvi., 1897, p. 185.

Nervus Intermedius of Wrisberg.—Between the facial and the auditory nerve, at the floor of the cranial cavity, there is a slender fasciculus, known as the *portio intermedia*. The fibres of this nerve are remarkably small in calibre. They appear to arise from the cells of the geniculate ganglion and run centrally into the bulb, alongside the fibres of the auditory nerve, to join a nucleus closely connected with that of the glosso-pharyngeal. Other fibres run peripherally, some into the great superficial petrosal nerve, others into the trunk of the facial, probably in the chorda tympani. The functions of this nerve are obscure. It is possible that it conveys some taste impulses upwards to the brain, by way of the glosso-pharyngeal nucleus (Fig. 54, p. 124). And it appears to contain also efferent fibres which join the submaxillary ganglion. Clinically we do not meet with isolated lesions of the nerve, so that its functions are less precisely known than those of any other cranial nerve.

No case of isolated palsy of the **Ninth or Glosso-pharyngeal Nerve** has yet been observed in man, so that its exact functions are not precisely determined. We know that it supplies taste-fibres to the posterior third of the tongue and to the soft palate. It probably also supplies the taste buds which exist in the epiglottis and on the arytenoid cartilage. But, as we have already seen, it is possible that some of these taste-fibres leave the glosso-pharyngeal at the petrous ganglion, and reach the brain through the fifth nerve (Fig. 48, p. 115). The glosso-pharyngeal is also a nerve of common sensation for the back of the tongue, part of the soft palate and upper part of the pharynx, whilst it has motor fibres for the middle constrictor of the pharynx and for the stylopharyngeus.

Paralysis of the nerve causes anæsthesia of the back of the tongue and pharynx, difficulty in swallowing and deficient taste in the posterior third of the tongue. In animals where this nerve has been divided experimentally, the pharynx and œsophagus remain tonically contracted, owing to paralysis of the inhibitory fibres contained in the glosso-pharyngeal.

The **Tenth Nerve, Vagus, or Pneumogastric Nerve,** according to modern nomenclature, is held to include those roots which used to be called the "bulbar part of the spinal accessory." Nowadays the term "spinal accessory" is limited to the spinal part of the accessorius which arises from an entirely separate nucleus, whereas the old "bulbar part" is derived from, and belongs to, a continuation of the vagus nucleus (nucleus ambiguus) in the medulla.

The vagus has a most extensive distribution. It supplies the pharynx, larynx, œsophagus, heart, lungs, stomach, and partly even the intestines and spleen. By its auricular branch it also supplies part of the skin of the outer ear. Its pulmonary fibres are motor for the bronchial muscles and sensory for the respiratory passages. The vagus is both motor and sensory for the œsophagus, sensory for the stomach, and partly motor for the stomach and intestines. Its lowest roots of origin are those which are of the greatest diagnostic importance, for they contain motor fibres for the levator palati and the larynx, together with inhibitory fibres for the heart. With the exception of the crico-thyroid muscle, which is innervated by the superior laryngeal branch, all the laryngeal muscles are supplied through the inferior or recurrent laryngeal nerve.

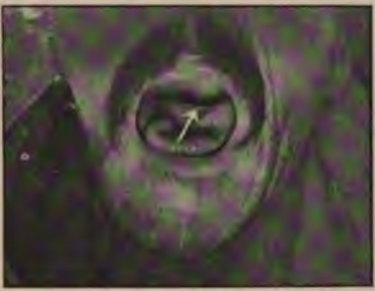

FIG. 57.—Paralysis of the right side of the palate. The patient is saying "Ah," and the palate is pulled up towards the left side. The arrow points to the centre of the uvula.

The symptoms of vagus paralysis vary according to the site of the lesion. Intra-cranial lesions may affect all its roots of origin, or may attack the upper or the lower roots alone. In the latter case there is often a concomitant affection of the adjacent hypoglossal nerve.

If the whole of one vagus trunk be affected, there is unilateral paralysis of the palate and larynx, together with anæsthesia of the larynx on the affected side. To recognise a unilateral paralysis of the palate we watch the movement of its median raphé when the patient utters a long "Ah." Normally the raphé rises straight up. But if one side of the palate be paralysed, the healthy side alone pulls upwards and the raphé deviates to the sound side (Fig. 57).

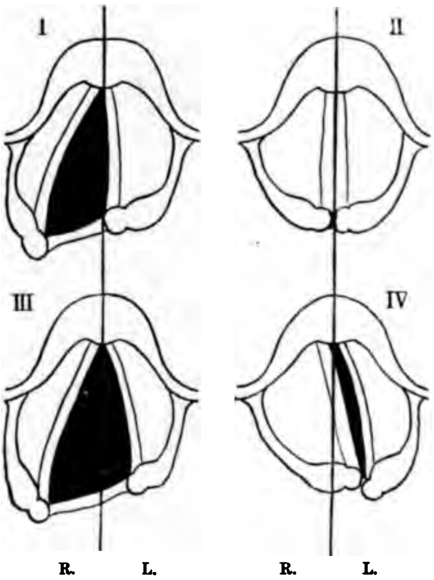

FIG. 58.—Organic laryngeal paralyses (Barwell).

I. Left abductor paralysis, during inspiration.
II. Left abductor paralysis, during phonation.
III. Left recurrent laryngeal paralysis, during inspiration.
IV. Left recurrent laryngeal paralysis, during phonation.

If both vagi be paralysed, there is tachycardia and irregularity of the heart, from paralysis of the cardio-inhibitory fibres. There is also slowness and irregularity of respiration. These do not occur in unilateral vagus palsy. Gastric symptoms have also been observed even in unilateral cases, such as gastric dilatation, vomiting, gastric pain, and loss of the sensations of hunger and thirst.

Of all these symptoms, the most constant and easiest to recognise are the affection of the soft palate and the laryngeal palsy. Paralysis of the recurrent laryngeal nerve may occur alone. The commonest cause is aortic aneurism, which frequently compresses the nerve on the left side. Mediastinal growths may also compress it, or its paralysis may even be the result of mitral stenosis, whereby the left auricle may compress the nerve directly against the pulmonary artery, or the auricle may force the left bronchus upwards and compresses the nerve against the aortic arch.[1] In *recurrent laryngeal paralysis* the vocal cord on the affected side is immobile, fixed in the cadaveric position, *i.e.* midway between abduction and adduction, and the voice is generally hoarse though not absent, since during phonation the healthy cord can cross the middle line to meet the paralysed one. If *both recurrent laryngeals* be paralysed, both vocal cords are motionless and in the cadaveric position, and phonation is impossible since the cords cannot be brought together. There is no stridor except on deep inspiration.

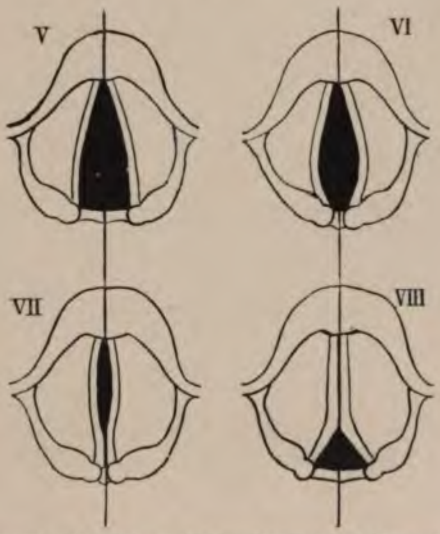

FIG. 59.—Functional laryngeal paralyses (Barwell).

V. Adductor paresis—all the adductors affected—phonation.
VI. Adductor paresis—arytenoideus unaffected—phonation.
VII. Paralysis of the thyro-arytenoidei—phonation.
VIII. Paralysis of the arytenoideus—phonation.

It is here convenient to recall some of the chief diagnostic features of laryngeal palsies (Figs. 58 and 59.) Abductor palsy, unilateral or bilateral, is always organic, and is often the earliest sign of a commencing recurrent laryngeal nerve affection. In *unilateral abductor palsy* the voice is unaffected, but on laryngoscopic examination the paralysed cord is seen to be immobile during inspiration, not moving outwards like its healthy fellow. On phonation the cords meet normally. In *bilateral abductor palsy* the voice is also unaffected, since both cords come together on phonation. But since they do not move outwards during inspiration, but on the contrary are sucked together, inspiration is laboured and stridulous, and the patient is in danger, since the slightest swelling of the cords may completely block the glottis. *Paralysis of the internal thyro-arytenoid muscle*, which occurs in some cases of early bulbar paralysis, is characterised by an oval instead of a linear appearance of the glottis on attempted phonation, owing to loss of the support of these muscles. The voice is

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[1] Frischauer, *Wiener Klin. Wochenschrift*, Dec. 28, 1905.

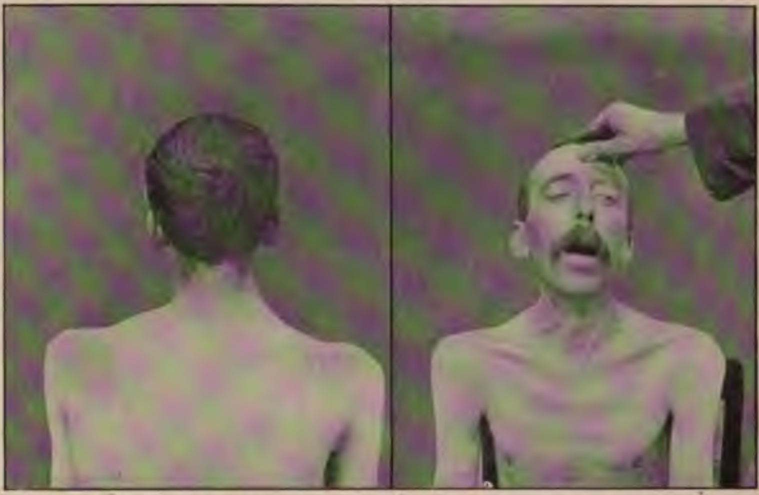

FIG. 60. FIG. 61.

Paralysis of right spinal accessory nerve.

Fig. 60 shows the downward and outward displacement of the right scapula. Observe alteration in the lateral outlines of the neck. On the paralysed side the outline is formed by the levator anguli scapulæ; on the normal side by the trapezius.

Fig. 61 shows the patient attempting to depress the head against resistance. Observe the absence of the right sterno-mastoid, the right omo-hyoid being now subcutaneous.

therefore hoarse, but abduction and adduction are otherwise unaffected. *Adductor paralysis* is always bilateral and generally hysterical. It is common in hysterical aphonia. The patient loses her voice, often suddenly, and talks in a whisper. There is no stridor, and on inspiration the cords move normally outwards. But on attempted phonation they do not reach the middle line. The condition often disappears suddenly, sometimes as a result of the process of laryngoscopy, or of the application of strong faradic shocks to the larynx.

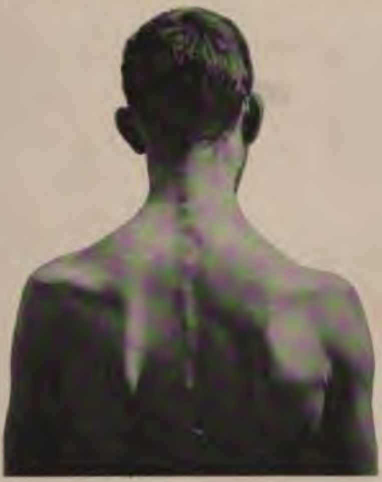

FIG. 62.—Paralysis of right spinal accessory nerve, showing downward and outward displacement of scapula. Owing to absence of the trapezius, the rhomboid muscles on the right side have become subcutaneous.

The **Eleventh or Spinal Accessory Nerve** is distributed to the sterno-mastoid and to part of the trapezius. It is exclusively a motor nerve. When it is paralysed, there is paralysis and atrophy of the sterno-mastoid, which no longer stands out on rotation of the head to the opposite shoulder, nor on depressing the head against resistance (Figs. 60 and 61). The paralysis of the trapezius varies in degree according to the extent to which the muscle is supplied by the spinal accessory and cervical plexus respectively. Ordinarily the uppermost fibres of the trapezius are innervated by the spinal accessory, whilst the middle fibres of the muscle are supplied by the third and fourth cervical nerves, and the lowest fibres of all are supplied by the spinal accessory. When the trapezius is paralysed, the outline of the neck is altered owing to the levator anguli scapulæ having become subcutaneous, and there is a characteristic deformity of the angle of the shoulder resulting from the displacement of the scapula (Fig. 62). The scapula on the paralysed side is displaced downwards and outwards, and rotated outwards, so that its inner border instead of being parallel with the spine, is inclined from below upwards and outwards. When the patient braces the shoulders back, the scapula is imperfectly approximated to the middle line, and the rhomboids on the paralysed side are visible subcutaneously (Figs. 63 and 64).

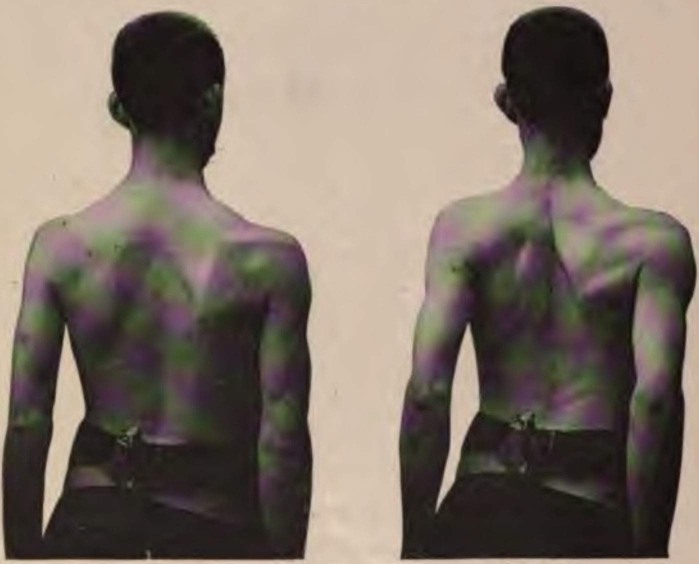

FIG. 63. FIG. 64.

Paralysis of right spinal accessory nerve.

Fig. 63.—Showing position at rest.
Fig. 64.—Showing position on adduction of scapulæ.

The **Twelfth or Hypoglossal Nerve** is also entirely motor in function. Immediately after its exit from the skull it receives amongst other connections a small communicating branch from the cervical sympathetic. The hypoglossal supplies all the intrinsic muscles of the corresponding half of the tongue. Outside the skull it is joined by branches from the first and second cervical nerves, and from these two nerves the depressors of the hyoid bone are supplied, through the *descendens hypoglossi*.

The symptoms of hypoglossal paralysis are very characteristic (Fig. 65). The corresponding half of the tongue is atrophied and wrinkled. When the tongue is protruded the healthy side causes the tip to be pushed round to the paralysed side in a sickle-shaped curve. If the nerve is divided suddenly, by accident or operation, the patient feels for a few days as if the paralysed half of the tongue were a foreign body, interfering with articulation and mastication. But this sensation soon passes off as the patient becomes accustomed to his hypoglossal palsy.

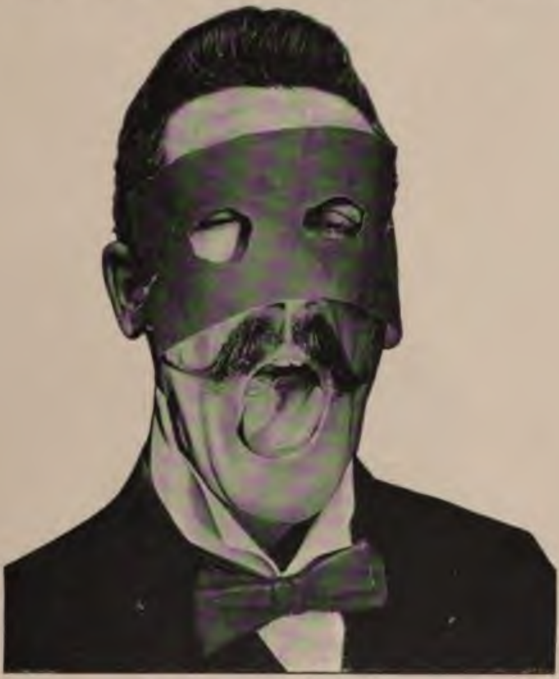

FIG. 65.—Paralysis of right hypoglossal nerve from a stab-wound below the jaw. Showing atrophy of corresponding half of tongue and marked deviation to the right when protruded.

If the lesion of the hypoglossal be extra-cranial, after it has received the small communicating branch from the cervical sympathetic, we may sometimes observe vaso-motor changes in the atrophied side of the tongue. In two of my cases where the twelfth was divided intentionally for the purpose of facio-hypoglossal anastomosis, the tongue was pale on the paralysed side.

The **hypoglossal nucleus,** as we have already mentioned, gives fibres to the facial nerve within the medulla, which supply the orbicularis oris. Hence in lesions of the hypoglossal nucleus, the muscles of the lips are paralysed together with those of the tongue. This weakness of lips and tongue in nuclear lesions is generally bilateral, owing to the close proximity of the hypoglossal nuclei to the middle line.

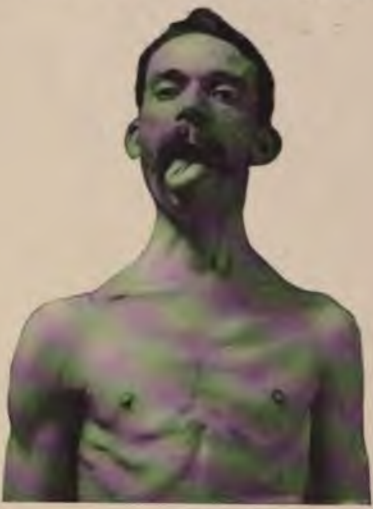

FIG. 66.—Paralysis of spinal accessory and hypoglossal on right side, showing downward and outward displacement of right shoulder, also atrophy of right half of tongue with deviation to the right on protrusion.

Intra-cranial lesions of the hypoglossal nerve, *e.g.* from a patch of syphilitic meningitis, very frequently involve other cranial nerves in the neighbourhood. One of the commonest multiple palsies is that which produces hemiatrophy of the tongue, paralysis of the trapezius and sterno-mastoid, and paralysis of the vocal cord and soft palate, all on the same side (Fig. 66). This is the result of a lesion implicating the twelfth, the eleventh, and the lowest roots of the tenth nerve.

LECTURE XI

PAIN AND OTHER ABNORMAL SUBJECTIVE SENSATIONS

THERE is, perhaps, no symptom for which we are more frequently consulted than that of pain And for its satisfactory treatment the underlying cause must first be determined—sometimes no easy matter.

With the exception of cases where pain arises in a healthy individual from some unduly strong stimulus, pain is always pathological. The vast majority of pains are due to irritation of some peripheral sensory nerve or of a posterior root. Less commonly pain may be due to abnormal sensitiveness of the cortical centres, and is functional in origin. Stimulation of the surface of the brain gives rise to no pain, but the meninges are exquisitely sensitive, the cerebral membranes being innervated by the trigeminal nerve. Intra-cranial diseases therefore probably cause pain chiefly through the intermediation of the sensitive meninges. It is possible, however, that the pain in some cases of syringomyelia may be due to actual distension of the syringomyelic cavity by the fluid within, though against this view is the fact that pain is a late phenomenon in this disease.

Spontaneous sensations of discomfort vary in degree and in kind. Milder varieties, not amounting to actual pain, are classed as **dysæsthesiæ.** They include such symptoms as spontaneous tingling, " pins and needles," dulness, itching, flushing, &c., whilst among the more severe varieties are the intense and agonising pains of tic douloureux, angina pectoris, renal or biliary colic, or the lightning-pains of tabes.

In the diagnosis of the cause of any particular dysæsthesia or pain, there is one invariable rule which we should always follow, namely, to make a careful local examination of the part of the body to which the abnormal sensation is referred. Only in this way can we escape gross errors, such, for example, as that of mistaking the pain of herpes zoster for that of pleurisy. In every local pain we should first search for a local cause, whether in the skin, muscles, bones, joints, glands, or other subjacent structures. Pain due to local disease is usually more or less continuous, and accompanied by objective phenomena such as redness of the skin, swelling or tenderness of the diseased tissues, rigidity of joints, and so on. The painful cramps of tetanus, rabies, and strychnia-poisoning are easily recognised and need not be further described.

Some pains are generalised all over the body, for example, the pains of acute illnesses such as influenza, smallpox, and other fevers of rapid onset. In such cases the elevation of temperature, the presence, perhaps, of a rash, and usually the occurrence of other similar cases in epidemic form, all help us in the diagnosis.

Most cases of pain or discomfort, however, are localised to some definite area. And therefore for diagnostic purposes the most convenient plan of studying pains and other subjective sensations is by considering the different parts of the body in turn.

Headache.—Cephalalgia.—A diagnosis of "headache" alone is never sufficient. Headache is not a disease, but merely a symptom.

When a patient complains of headache, local causes should first of all be excluded. Amongst the commonest *local extra-cranial* causes we should bear in mind rheumatic affection of the occipitofrontalis muscle, with its diffuse tenderness on brushing the hair, and with aggravation of pain on movement of the scalp. Erysipelas is accompanied by local redness, heat, and œdema, and by fever. Periostitis of the pericranium causes tenderness on pressure and is often syphilitic in origin, in which case the pain is commonly worse at nights.

Local causes in the *cranial bones* are most commonly due to inflammation of the mucous membrane lining the various air-cells, *e.g.* the mastoid, frontal, or ethmoidal cells, or even in an ordinary coryza. Here the previous history of the case, together with the local examination, will serve to indicate the cause. Less frequently caries of the bones or local exostoses may be present. Carious teeth often cause headache, referred especially to the temporal region.

Gross intra-cranial disease may produce intense headache, either from local affection of the meninges as in the various forms of meningitis (syphilitic, tuberculous, or pyogenic), or from general increase of intra-cranial pressure, as in diffuse meningitis, intra-cranial abscess, gumma, or tumours. Headaches of intra-cranial origin are often associated with the other classic signs of increased intra-cranial pressure, especially with vomiting and optic neuritis. In every case of obscure headache we should make a careful ophthalmoscopic examination. The position of the headache does not necessarily correspond with that of the tumour. Tumours of the posterior fossa produce the most severe headache.

Then there are the headaches which result from abnormalities of the intra-cranial circulation, especially from arterial hyperæmia, as in renal disease or in suppressed menstruation, or temporarily from inhaling nitrite of amyl or from excess of alcohol. Hyperæmia produces a throbbing pain, sometimes associated with giddiness, tinnitus, affection of vision, and a tendency to attacks of epistaxis. The venous hyperæmia of severe cardiac disease or of emphysema may also produce a headache, which is aggravated by coughing. The headache of anæmia is probably mainly toxic in origin, rather than due to actual deficiency in the supply of blood to the head.

Certain cases of hemiplegia are preceded, for days or even for weeks, by headache. This is especially common in *cerebral thrombosis*, where a premonitory headache is of considerable diagnostic value. The onset of a *cerebral hæmorrhage* is often coincident with a headache. Therefore if an elderly man complains of headaches and bleeding from the nose, we should be cautious in checking epistaxis which may act as a safety-valve, and may save him from an attack of cerebral hæmorrhage.

There is another group of headaches which are produced by peripheral irritation in various parts of the body. Thus, for example, visceral irritation may cause headache, as in ovarian or uterine diseases, or in the headaches from which so many women suffer at the menstrual periods, and frequently also throughout the menopause. In some people the swallowing of an ice causes sudden frontal headache, when the ice reaches the stomach. This is a typical "reflex" gastric headache. Every autumn we come across cases of "gun headache" amongst sportsmen, the result not simply of the auditory stimulus of the noise of the gun, but due partly to the shock of repeated recoils of the weapon. Ocular headaches are fairly frequent, sometimes the result of disease such as glaucoma or iritis, but much more commonly arising from errors of refraction, especially hypermetropia and astigmatism. Even apart from this, headache may result from prolonged eye-strain, *e.g.* after sight-seeing in picture-galleries, but it is commoner in people who have some error of refraction.

A large group of headaches are *toxic* in origin, the toxins altering the intra-cranial circulation. The onset of certain infective fevers is associated with headache, for example in smallpox and influenza (usually associated with pain in the back), in enteric fever associated with dyspeptic symptoms, in scarlatina, pneumonia, &c. In such cases the temperature, the characteristic rash, and the other signs and symptoms will guide us. Poisoning by toxic substances is also the main cause of the headache of dyspepsia, whether it be the familiar "Katzen-jammer"—the morning-headache and nausea following acute alcoholic excess; or the headache of chronic dyspepsia, especially when associated with constipation. Headaches also result from poisoning with carbonic oxide, carbonic acid, and other respiratory sewage in ill-ventilated rooms or at crowded meetings. Other poisons, again, are autogenetic in origin, as are the headaches of uræmia and diabetes.

Sunstroke causes acute diffuse headache. Severe cases are often accompanied by other symptoms such as delirium, hyperpyrexia and coma. Neurasthenia and exhaustion, whether from overwork, from excessive study or from other excesses, is often associated with headache, frontal, occipital, or circular (*douleur en casque*). The post-epileptic headache following a fit is sometimes severe and may be associated with vomiting.

Bilateral headaches are most commonly toxic. The headache of dyspepsia is usually referred to the frontal region, that of constipation to the occiput, whilst vertical headaches are most commonly due to anæmia or to debility. Some cases of concussion of the brain are followed by obstinate headaches, recurring for many months. These, I believe, are sometimes partly toxic in origin, a smaller amount of toxin being efficient in producing headache after concussion than in the case of a healthy individual, whilst in other instances the headaches occur on slight mental exertion, especially where the patient has been allowed to return to work prematurely. Hence the importance of complete physical and mental rest after a severe head injury, even in the absence of signs of an organic lesion.

Most of the headaches to which we have referred are bilateral or mesial in situation. Let us now consider a different group—the unilateral or circumscribed pains.

Sometimes these are due to local disease of the scalp, pericranium, or skull, in which case local examination will generally reveal the cause. Or they may result from intra-cranial disease, for example, meningitis, abscess, gumma or tumour. If the underlying disease be near the surface of the skull, the site of the pain sometimes corresponds with that of the disease. But this is by no means always the case. I remember a striking case of right-sided cerebellar tumour where the pain was confined to the left supraorbital region. The growth was diagnosed and successfully removed.

Some headaches are associated with great pallor of the face —apparently due to vaso-motor spasm of the cerebral vessels, often relieved by inhalation of amyl nitrite. Others are associated with arterial hyperæmia, throbbing arteries, and a flushed face. These are sometimes promptly relieved by compression of the carotid artery on the corresponding side. Such angio-neurotic headaches are rarely bilateral.

A very acute circumscribed variety of headache is known as the *clavus hystericus*, an agonising pain usually referred to the vertex, as if a nail were pressing into the brain. It occurs in certain cases of hysteria and neurasthenia. It is unassociated with any other evidences of intra-cranial disease, and its very intensity leads us to be suspicious as to its true nature.

The pain of sick-headache or *migraine* (hemicrania), with its paroxysmal attacks occurring at intervals of days or weeks, the patient during the intervals being free from headache, is usually easy of recognition. This headache generally culminates in vomiting, and is sometimes preceded by visual phenomena in the form of a scintillating scotoma, consisting of a blind area in one half of the visual field, on the side opposite to that of the unilateral headache, the blind area being bounded at its periphery by a zig-zag coloured spectrum. This scotoma not infrequently developes into a temporary total hemianopia. *Migraine ophthalmoplegique* is a rarer variety, in which, in addition to hemicranial pain, there is transient paralysis of the third nerve on the same side as the headache, with ptosis, external strabismus, mydriasis, &c.

We now pass to pains in the region of the trigeminal nerve. Of these the most severe is *tic douloureux*, the most appalling form of neuralgia. It rarely attacks all three divisions of the nerve, but is usually confined to one (especially the supraorbital division), or it may attack two adjacent divisions. The disease is hardly ever bilateral except in cases of diabetes. The patient has paroxysms of intolerable agonising pain in the area of the affected division. During the attack, the face is often thrown into strong involuntary tonic spasms on the affected side, there is excessive lachrymation, and sometimes secretion of nasal mucus and saliva, all on the affected side. Not only do paroxysms occur spontaneously, but the slightest stimuli often suffice to induce an attack, and therefore the patient avoids chewing food on the affected side. I have even known cases in which it was impossible for the patient to wash his face for weeks at a time, lest an attack should be thereby precipitated.

Apart from these, there are numerous varieties of more localised paroxysmal neuralgic pains referred to individual branches of the trigeminal nerve, and associated with "tender points" over their foramina of exit. In such local neuralgias, and in tic douloureux itself, we should always search carefully for some local exciting cause in the mouth, nose, ear, or eye. A carious or inflamed tooth may cause neuralgia in the whole of the corresponding division of the fifth nerve, and the dental surgeon by extracting it will relieve the condition. But we must beware of extracting sound teeth simply because the patient refers neuralgic pain to them. Sometimes an abnormally-placed tooth, though healthy in itself, may cause neuralgia. Thus a lady of fifty-eight had a constant burning pain along the right side of her tongue for eight years, together with a feeling of numbness in the area of the second division of the fifth nerve. This had been vainly treated by numerous drugs, whilst all the time the real exciting cause lay in an imperfectly-erupted lower wisdom-tooth on the corresponding side, the date of the pain coinciding with the first appearance of the tooth. Antral disease, nasal polypi, and other local lesions may all cause localised neuralgias. If, in addition, the patient be anæmic or gouty, the tendency to neuralgia is increased. But anæmia or gout alone will not cause a local neuralgia. Some local determining cause must also be present, though it is sometimes difficult to discover. Syphilitic basal meningitis or gumma, implicating the fifth nerve in the floor of the skull, may cause trigeminal neuralgia. Such cases usually show other evidences of intra-cranial disease, and we should look for signs of involvement of the motor root (see page 118). If such lesions last any considerable time, they tend to produce some anæsthesia of the affected nerve-area. Localised anæsthesia will suggest an organic neuritis rather than a mere neuralgia.

Let us now consider the various pains which may be met with in the **trunk**. Firstly, there are the various root-pains, due to irritation of the corresponding posterior root or roots in the affected area. Sometimes such root irritation is due to disease of the spinal meninges (tubercle, syphilis, or tumour), to osteo-arthritis, caries or tumours of the spinal column, or to intra-thoracic aneurisms and new growths. Bone pains in the spine are associated with local tenderness and rigidity. If we see a patient supporting his head with both hands owing to pain in the neck, this is almost pathognomonic of disease of the cervical vertebræ. Root-pain commencing unilaterally and later becoming bilateral is practically pathognomonic of a tumour of the spinal meninges. These pains when due to organic lesions of the roots, are not infrequently associated with a degree of hyperæsthesia or anæsthesia of the painful area—*anæsthesia dolorosa*. If the spinal cord be compressed or infiltrated by the same lesion as that which implicates the posterior roots, we have evidence of sensory or motor paralysis of the parts below the lesion, with the usual changes in the reflexes, &c. The *girdle-pains of tabes* are due to affection of the corresponding posterior roots. Tabetic girdle-pains vary in degree from the sensation of a narrow constricting cord to one of a broad cuirass enveloping a large part of the trunk. Such a cuirass often feels incomplete either in front or behind (Fig. 71, p. 171). Another root pain is that associated with herpes zoster, which is a disease of the posterior root-ganglion. Herpetic pain is practically always unilateral, and may either precede or succeed the eruption of the herpetic vesicles. It may last for months after the vesicles have disappeared. The pain of herpes is often so sharp that it may be mistaken for that of pleurisy, from which it is distinguished by auscultation. Pleurodynia is a pain in one or more intercostal spaces, due to a "rheumatic" myalgia of some of the intercostal muscles. It somewhat resembles the pain of pleurisy, but is easily distinguished by the local tenderness on digital pressure and by the absence of friction-sounds on auscultation. There is also a very common trunk-pain which we meet with in people who are neurasthenic or debilitated. It is a deep boring pain, usually below the inferior angle of one or other scapula. It is more diffuse and less superficial than the pain of herpes, and it has none of the physical signs of pleurisy or pleurodynia. The pain of traumatic neurasthenia, especially after a railway or other accident ("railway spine"), may simulate that of organic spinal lesions, especially when a functional paraplegia coexists. But the diagnosis can usually be made by noting the excessive hyperæsthesia of the spine, the absence of signs of organic disease and the presence of various hysterical "stigmata" (see later, p. 312). Mammary neuralgia or mastodynia also occurs in neurasthenic patients, and must be distinguished by means of physical examination from disease of the gland.

We have also to bear in mind the various *referred pains* which may occur in visceral diseases. Thus, for example, in pericarditis there may be precordial or epigastric pain. In aortitis pain may be referred to the upper three intercostal spaces on the right side. Physical examination will clear up the diagnosis in cases of aneurism and of mediastinal growths. One of the most severe of all trunk pains is the well-known *angina pectoris.* This is a paroxysmal suffocative pain, or feeling of intolerable oppression in the region of the heart, often radiating down one or both arms, but especially down the left arm. It is commonest in male patients at or after middle life, and is usually, though not invariably, associated with evidence of vascular degeneration. It has to be distinguished from toxic or neuralgic angina, so-called "pseudo-angina," a similar but much less serious affection, met with most commonly in young girls, in women who have been lactating too long, or in patients before middle life who smoke tobacco or drink tea to excess.

Irritation of the sensory nerves at the cardiac end of the stomach, by abnormal acids or other irritants, may cause burning pain, usually referred to the seventh left chondro-sternal junction, and sometimes also to the left interscapular region. Such gastric pains are associated with other dyspeptic symptoms to which we need not here refer further. As regards the situation of referred pains in diseases of various parts of the gastro-intestinal tract, it is useful to bear in mind Mackenzie's rule,[1] that pain due to affections of the digestive tract is referred across the middle line of the abdomen, in regularly descending areas as we pass from the stomach towards the large intestine. Thus the epigastrium is the region for gastric pains (disease at the cardiac end causing pain higher up than pyloric affections), the umbilical area is the region for pains of the small intestine, the hypogastric area the site of pains due to the large intestine. A striking experimental corroboration of this can be obtained at any time by taking a sharp purgative drug. When the familiar colicky pains appear, they are felt first in the region of the umbilicus, but soon they descend lower and lower, and when they arrive close above the pubes, the call for evacuation of the bowel becomes " urgent and imperative."

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[1] *Brain*, 1901, vol. xxv. p. 373.

To discuss fully the various causes of acute abdominal pain would require many chapters of description. We have to bear in mind not only diseases of the gastro-intestinal tract, in the form of catarrh, ulcer, muscular spasm, &c., but also perforations of various hollow viscera, the stomach, gall-bladder, intestines (including the vermiform appendix), rupture of a pyo-salpynx or of a tubal pregnancy, biliary or renal colic, acute pancreatitis, torsion of an ovarian pedicle, &c. In every instance, not only should we carefully examine the whole abdomen, but we should, if necessary, examine the pelvis, per rectum or per vaginam, and investigate the urine and dejecta.

Apart from acute renal colic, a floating kidney is a fairly common cause of diffuse abdominal pain, especially in poorly-nourished women with lax abdominal walls. Here again, local examination of the abdomen will reveal the cause.

Before leaving the subject of abdominal pains, we must not fail to recall the familiar *crises* of tabes dorsalis :—gastric crises associated with pain and vomiting, intestinal crises associated with colic and diarrhœa, bladder crises, &c. All these may closely simulate the pain of acute abdominal disease. But the history of the case, together with an investigation of the pupils, knee- and ankle-jerks, and the other phenomena of tabes, will usually save us from error.

Lumbago, or pain in the lumbar muscles, is usually easily recognised. The pain is intensified by active muscular contraction and also by passive stretching, caused for instance by the stooping posture. It is also associated with tenderness on pressure. Lumbo-abdominal neuralgia, on the other hand, is a diffuse pain, not confined to the lumbar muscles but spreading forwards from the lumbar region to the front of the abdomen, and sometimes to the groin, genitals or gluteal regions. It is paroxysmal, and during the paroxysm there may be cramp-like spasms of the abdominal muscles or of the cremaster. It is associated with the " tender points " of a true neuralgia, these being situated over the vertebral spines, the iliac crests, the linea alba, inguinal canal, scrotum or labium. Sacral pain is often due to uterine disease, as in the familiar uterine dysmenorrhœa or the well-known pains of labour.

Coccygodynia or neuralgic pain in the region of the coccyx, is practically confined to the female sex. The pain may be spontaneous, or it may be induced by sitting or walking or by the contraction of any of the muscles attached to the coccyx, *e.g.* during defæcation. It is often associated with local tenderness. Before making a diagnosis of mere neuralgia, local disease of the coccyx or of adjacent structures must always be excluded by local examination.

Pains in the Limbs.—Brachial neuralgia, generally a unilateral affection, is referred, as a rule, to the whole area of distribution of the plexus, namely to the lower part of the neck, the shoulder and the whole upper limb. It is rare to meet with neuralgia confined to an individual nerve-area, such as that of the median or ulnar, except in cases of local injury or disease of the nerve-trunks. The pain of brachial neuralgia is aggravated by movement of the limb, which feels heavy and numb, though there is no paralysis. The " tender points " are over the nerve trunks, such as the musculo-spiral, median or ulnar nerves.

If anæsthesia or trophic changes be superadded, we probably have to do with structural changes in the nerve-trunk, that is, with a neuritis, not a mere neuralgia. In every case of brachial neuralgia we must carefully examine the nerve-trunks in their entire course, to exclude the possibility of organic lesions compressing or infiltrating the nerves. In addition to pain, brachial neuritis often produces weakness and atrophy of the corresponding muscles, and impairment or perversion of cutaneous sensation.

Analogous to brachial neuralgia and neuritis in the upper limb, we have in the lower limb *sciatica*, a term which includes sciatic neuralgia and sciatic neuritis. In every case of so-called sciatica we have to decide which of these two is present. In sciatic neuralgia there is usually a dull aching pain more or less constant, in the back of the thigh, with occasional paroxysms of darting or boring pain, generally from above downwards, along the course of the sciatic nerve. Any movement of the limb whereby the nerve is made tense, or any local pressure as from sitting on a hard chair, brings on a paroxysm. Therefore the patient habitually keeps the hip and knee slightly flexed on the affected side, so as to relax the nerve. And it is not uncommon to find a slight degree of scoliosis in the lumbar region, the concavity being towards the sound side. The " tender points " are at the posterior iliac spine, the sciatic notch, the popliteal space, the peroneal nerve below the head of the fibula, and behind the malleoli. Passive stretching of the nerve increases the pain, for example by flexion of the hip with the knee extended. Blunting of sensation in the peroneal or posterior tibial area is uncommon and indicates an organic neuritis or perineuritis, as also do any alterations in the electrical reactions of the muscles, or any considerable degree of muscular atrophy. In sciatic neuritis the temperature of the limb is generally lower than on the healthy side. The ankle-jerks should always be tested on both sides. In sciatic neuritis the jerk may be diminished or lost, whereas in neuralgia it remains normal. In both affections we may observe exaggeration of the cremasteric reflex on the affected side.

Meralgia paræsthetica is a variety of neuralgia occurring in the area of distribution of the external cutaneous nerve of the thigh. It consists in paræsthesia or actual pain in the outer aspect of one thigh. The pain is often induced by standing or walking, possibly owing to stretching of the fascia lata. It is sometimes associated with flat-foot on the same side. Here, as in brachial or sciatic pain, the presence of an area of impaired sensation would indicate a neuritis rather than a neuralgia.

In rare cases we may find neuralgia in the area of the anterior crural or of the obturator nerve, and this may be symptomatic of an intra-pelvic tumour, or of an obturator hernia.

The pains of brachial or sciatic neuralgia and neuritis, and of meralgia paræsthetica, are unilateral. Let us now consider the bilateral pains which may be met with in the limbs.

Bilateral pains should always suggest to our minds either a toxic cause attacking the peripheral nervous structures of both limbs, or some central disease of the spinal meninges affecting the posterior roots bilaterally, or again some angio-neurotic condition such as Raynaud's disease, erythromelalgia, or intermittent limp.

The *lightning pains* of tabes may be unilateral or bilateral. They are commoner in the legs than in the arms, since tabes is a disease which commonly begins in the posterior root-fibres of the lumbo-sacral region. These pains are variously described by the patient as stabbing, burning, tearing, or bursting, and are commonly associated with local hyperæsthesia of the skin. They are often mistaken for rheumatic pains, and all the more so inasmuch as they often coincide with changes in the weather.

Tight "tourniquet" pains around the lower limbs, in broad zones rather than narrow, are sometimes an early and persistent symptom in disease of the lumbo-sacral region of the cord. They are due to irritation of the posterior roots, and may also occur in tabes.

The *root-pains* of tumour or inflammation of the spinal meninges, or of spinal caries, are more or less constant, with paroxysmal exacerbations. Inflammatory affections of the meninges are usually bilateral from the outset, with corresponding bilateral pains. But in cases of meningeal tumour the pains are generally unilateral at first, and become bilateral as the disease spreads to the opposite side. The level of the pains in meningeal disease varies with the level of the affected posterior roots. Thus in cervical meningitis, tumour or caries, there is pain in the neck, spreading down one or both arms along the corresponding root-areas; in dorsal cases the pain is around the trunk, and in lumbar or sacral cases it is in various parts of the lower limbs. Meningeal pain is often associated with local hyperæsthesia corresponding to the uppermost roots affected, and with tonic spasms of the muscles at that level.

If the meningeal lesion affects the cord within, whether by compression or by infiltration, there will be in addition to root pains the other signs of organic cord disease—so-called *araplegia dolorosa,* with its anæsthesia, motor weakness and alterations of reflexes below the level of the lesion. Inflammatory, tuberculous and syphilitic affections of the meninges are always associated with cellular changes in the cerebro-spinal fluid (see p. 353).

Affections of the peripheral nerves may also produce pain. Thus in peripheral neuritis there is not only pain, with hyperæsthesia of the skin of the feet and hands, but there is intense muscular tenderness on pressure, together with a degree of tactile anæsthesia, and in severe cases muscular paralysis and muscular atrophy, accompanied by the reactions of degeneration.

Pains localised in single nerve-areas should always lead us to examine the nerve-trunk in its entire extent. *Tubercula dolorosa* are multiple growths (usually neuro-fibromata) in the connective tissue of the nerve-trunks, many of them forming little subcutaneous nodules easily palpable and exquisitely tender, others less accessible in the deeper nerve-trunks, causing referred pains in the particular nerve areas. If these growths not merely irritate but interrupt the nerve-fibres within the nerve-trunks, there may in rare cases be areas of anæsthesia. Clinically we seldom find motor paralysis from such growths, except as a result of pressure on the spinal cord or base of the brain by a neuroma on one of the spinal or cranial nerve-roots

It is convenient here to refer to erythromelalgia, where there are cyanosis and pain in one or both feet in the dependent posture, relieved by elevating the limb, to Raynaud's disease, which may be associated not only with local pallor, cyanosis or gangrene, but also with subjective sensations of tingling or pain, and to intermittent limp, when the patient after a few steps becomes unable to walk farther, owing to intolerable pain in the muscles of the leg. To these conditions we shall return later, when studying the nervous affections of the vascular system.

Finally, we should refer to the group of pains met with in hysteria. These are more often areas of hyperæsthesia than of spontaneous pain. They are specially common in the neighbourhood of joints, whose slightest movement causes intense pain. But the history of the case, in which there has often been a preceding injury, the absence of signs of structural disease local or central, and the presence of other hysterical stigmata, will aid us in our diagnosis. It may be necessary to give a general anæsthetic, in order to eliminate gross local organic disease.

LECTURE XII

ABNORMALITIES OF SENSATION: HYPERÆSTHESIA, PARÆSTHESIA, ANÆSTHESIA

We have already considered the anatomical course of the chief sensory paths from the periphery to the perceiving centres in the brain (Lecture I.). Let us now proceed to consider the methods of clinical investigation of the various forms of sensation.

All parts of the surface of the body are not equally sensitive. Thus the tip of the tongue, the lips, the finger-tips, in the order mentioned, are the most sensitive to cutaneous impression, whilst other parts such as the dorsal aspect of the trunk, the upper arm, and the calf of the leg, are least sensitive. These differences depend on various factors, such as thickness of epithelium, relative abundance of sensory end-organs, &c., into which we need not enter more minutely here.

All sorts of ingenious apparatus have been devised for the accurate measurement of minute differences in sensibility to touch, pressure, pain, temperature, and so on. But for clinical diagnosis, we should avoid complicated apparatus and content ourselves with the simplest possible methods which, whilst accurate enough for practical purposes, do not impose too great a strain on the patient's attention nor demand too high a degree of intelligence on his part.

The most important varieties of sensory stimuli which we employ in testing a patient's sensory functions are light touches, pin-pricks, cold and hot objects, all of which refer to *cutaneous sensations*. We have also to consider other sensations, such as *joint-sensation* (or sense of position on passive movement), *active muscle-sensation* (kinæsthetic sense, or sense of active muscular contraction) and, lastly, the *vibration sensation* produced when a sounding tuning-fork is placed over the subcutaneous surface of a bone or upon a finger-nail. There are other varieties of sensory stimuli, such as electro-cutaneous sensibility (which is generally, but not always, parallel in intensity with the pain-sense), and there is the sensation of pressure and appreciation of differences of pressure, &c. But these, whilst physiologically interesting, are of minor clinical value.

There is perhaps no better criterion of practical neurological skill than the accuracy with which an observer can map out areas of diminution or loss of sensation on the one hand, or of perverted or exalted sensation on the other. Both experience and patience are required, in order to obtain trustworthy results.

Inasmuch as we are largely dependent on the intelligence and goodwill of our patient for accurate answers, we must as far as possible try to eliminate all distracting outside factors. Therefore we direct the patient to close his eyes when we are testing sensation, so that his attention may not be diverted by watching what is being done. We must also be careful not to weary a patient by too prolonged examination, lest as he gets tired or impatient his answers become inaccurate. The simpler our methods of examination, the better are our results likely to be. We have also to contend with wide variations in the intelligence of different patients, in their education, and in their attentiveness; this latter may be modified by pain, by anxiety, or by psychical deficiencies. Sometimes we have to deal with deliberate attempts on the part of the patient to mislead us. Fortunately, patients who simulate disease generally make blunders so gross as to prevent an erroneous diagnosis on the part of a careful observer.

Clinical Investigation of Sensation.—At the start, the patient's eyes should be closed, or some object should be interposed between his eyes and that part of the body which is being tested. We then proceed to test the various *cutaneous sensations*—touch, pain and temperature, separately and in turn.

Touch is tested by means of some soft light object, such as a tuft of cotton-wool, a feather, or a gentle touch with the finger, or by gentle pulling or stroking of the hairs. Loss of sensation in the hairs is called "tricho-anæsthesia." *Pain* is tested by a sharp needle; *cold* by blowing on the skin, or by a cold object such as a metal spoon or a test-tube containing ice-cold water; *heat* by breathing on the skin or by a warm object such as a test-tube containing hot water.

Each variety of sensation should be examined separately, before passing on to the next kind of stimulus, and the results should be recorded on an outline-chart of the body. In mapping out areas of abnormal sensation, it is useful to have a skin pencil with which to mark on the patient's skin, before copying the result on our chart.

An important practical point in mapping out areas of anæsthesia, is to begin within the anæsthetic area, and to work towards the normal skin, not in the reverse direction. It is easier for a patient to recognise the moment when he first feels a sensation than for him to observe when he first loses it. On the other hand, in mapping out areas of hyperæsthesia or of paræsthesia, we should work from normal skin towards the hyper-æsthetic area, asking the patient to call out as soon as his sensation changes.

In setting about the examination of the sensory functions, we usually begin with that of touch. The patient's eyes being closed, we touch him lightly on both sides of the face simultaneously and observe not only whether he feels the touches, but whether they are equally distinct on both sides. We then touch symmetrical spots on the neck, shoulders, hands, trunk, and lower limbs. We next proceed to do the same with light needle-pricks, then with cold and with warm objects. If the patient has an area of diminished or altered sensibility, we generally discover it by this method. When we find an area of abnormality, we proceed to map it out carefully, making separate observations for touch, pain, and temperature, and noting whether the areas coincide or overlap.

Besides noting whether a patient feels a stimulus, for instance a tactile one, we should also notice whether he localises it accurately. This is accomplished by asking him to place his finger on the spot where he was touched, for instance, the dorsum of the hand. A normal individual can do this accurately to within a fraction of an inch. But in certain varieties of anæsthesia, the patient, whilst able to tell that he has been touched, makes an error of several inches in localisation. This is called "topo-anæsthesia," or, if it refer to pain, "topo-analgesia." Horsley[1] has shown that errors in the localisation of cutaneous impressions are, in cases of cortical lesions, always in a proximal direction, *i.e.* the patient refers the stimulus to a point higher up the limb. Sometimes the patient, when touched on one side of the body, feels the sensation at the corresponding spot on the opposite side. This is termed "allocheiria," and occurs only in certain cases of hysteria.

When testing pain, we sometimes find that though the patient correctly perceives and localises the stimulus, there is an abnormally long interval of time, perhaps amounting to several seconds, between the stimulus and the patient's perception of pain. This is called "delayed sensation," and is met with chiefly in cases of tabes.

So much for cutaneous sensations. But there are other forms of sensation which are of clinical importance. *Joint-sense* is tested by moving a joint passively into various positions backwards and forwards, then holding it fixed in a certain position, such as that of semi-flexion, and asking the patient to imitate exactly that position with the limb of the opposite side. It not infrequently happens that when a patient is in doubt as to the position of his joint, he begins to make slight voluntary movements of the joint before answering. These must not be permitted, since he thereby gains information as to the position of the limb, not from his joint-sense but through an entirely different sense, viz., the kinæsthetic sense or sense of active muscular contraction.

To test this *kinæsthetic sense,* we notice whether the patient, when raising his limbs, can detect differences in the weights of objects of similar size, for example a shilling and a sovereign, either placed in his hand, or hung in a sling over his hand or foot. For this purpose we sometimes employ a series of leather or wooden balls of equal size, loaded with different weights. Normally, according to Weber's law, a healthy individual should detect an increase of one-third in the weights of two successive objects. Tabes is the disease in which this sense of active muscular contraction is most markedly diminished, and where the joint-sense is notably impaired also. The loss of these two senses is probably the main factor in the production of tabetic ataxy.

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[1] *Brain*, 1906, p. 137.

We purposely avoid using the term "muscular sense," for several reasons. Firstly, it is ambiguous, since it has been used to include two entirely different senses:—joint-sense and kinæsthetic sense. Moreover, it might also be confounded with a third sense, the *sensibility of muscles to pressure* with the fingers. Normally such pressure, if moderate in degree, is painless; but in certain diseases, as in peripheral neuritis, in the various forms of myositis, and in the abdominal muscles superficial to an area of peritonitis, the muscles become exquisitely tender to the lightest pressure. On the other hand, it is common to find in tabes that severe compression of the muscles and tendons, for example of the leg muscles, and especially of the tendo Achillis, is painless. This muscle and tendon analgesia is often present in early stages of the disease.

It is sometimes of value to observe the patient's power of recognising, without seeing them, the forms of solid objects placed in his hand—so-called *stereognostic perception*. Normally a patient should be able to recognise familiar objects such as a key, a coin, or a chain. But in some cases the patient, whilst able to feel the presence of some object, cannot tell what it is, without seeing it. Such "astereognosis" may be due either to impairment of sensation from peripheral disease, or to defects in the cortical centres, as in certain cerebral lesions.

Lastly, there is the *vibration-sense* (pallæsthesia, or "osseous sense") described originally by Egger. This is tested by means of a low-pitched tuning-fork, which is set into vibration and placed upon the subcutaneous surface of a bone. In normal individuals a characteristic vibratile thrill is felt. But in certain diseases involving the posterior roots, such as tabes, or in transverse lesions of the spinal cord, the vibration-sense may be lost in the bones corresponding to the affected roots (Fig. 67). Loss of this sense may be the earliest form of anæsthesia in root-lesions as in tabes, where it may precede cutaneous anæsthesia. In Brown-Séquard paralysis vibration-sense is lost, together with the temperature sense, on the side opposite to that of the muscular paralysis (see Figs. 8 and 11). But the vibration-sense is not an exclusive property of bones, though bones are most strikingly sensitive; it can be perceived in other tissues, notably in the nails, which are closely connected with the periosteum, and even in the connective-tissues, though in them less intensely.

Excessive sensitiveness to normal stimuli is termed *hyperæsthesia*. Such hyperæsthesia is usually accompanied by a degree of discomfort or even pain, even though a stimulus be used which is ordinarily painless. The term *paræsthesia*, or perversion of sensation, signifies that an ordinary stimulus evokes an unusual sensation, as for example a feeling of tingling when the skin is touched, or a feeling of acute pain when moderate cold is applied. Under the head of paræsthesiæ we may also include such phenomena as multiple sensations (polyæsthesia), allocheiria, &c.

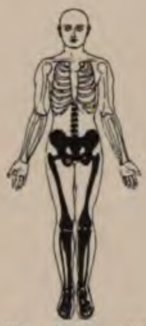

FIG. 67.—Tabes with loss of vibration-sense in bones of lower limbs, pelvis, lumbar, and lower dorsal vertebræ. The bones with loss of vibration-sense are shaded black.

Strictly speaking, diminution of sensation should be designated *hypo-æsthesia*, and the term anæsthesia should be reserved for total loss of sensation. It is usual, however, to speak of "slight," "moderate," and "total" anæsthesia. When only one form of cutaneous anæsthesia is referred to, we sometimes find it convenient to talk of *tactile anæsthesia*, of *analgesia* or loss of pain sense, and of *thermo-anæsthesia* or loss of temperature sense *Dissociated anæsthesia* is where some forms of sensation, such as tactile sense, are normal, whilst in the same area others, such as pain and temperature sense, are lost. This occurs especially in syrin omyelia and in the Brown-Séquard syndrome.

We speak of *hemi-anæsthesia* where one-half of the body, right or left, is affected, and of *para-anæsthesia* where both legs or both arms are affected owing to a lesion of the spinal cord or to a symmetrical affection of the posterior roots. We also speak of radial, ulnar, peroneal anæsthesia, &c., where the sensory loss corresponds to the distribution of a single peripheral nerve.

Hyperæsthesia.—Universal hyperæsthesia is rare. It is chiefly met with in hysteria, but also occurs in other affections, as in strychnia-poisoning, where the slightest touch may suffice to evoke a violent spasm. Hemi-hyperæsthesia is chiefly found in neurasthenic and hysterical patients and may be associated with other hysterical "stigmata." Thus I remember the case of a soldier with traumatic hysteria who had hemi-hyperæsthesia, accompanied by abnormal widening of the visual field and by increased acuity of smell, taste, and hearing, all on the hyperæsthetic side of the body.

Hyperæsthesia in more or less symmetrical root-areas of the trunk or limbs and due to irritation of the posterior roots, is not uncommon in diseases causing pressure on the spinal cord or its meninges, as in caries or tumours of the spine. Here the hyperæsthesia corresponds to the area supplied by the uppermost root involved, and is usually associated with anæsthesia and motor weakness in the parts below.

Odd irregular areas of hyperæsthesia are amongst the commonest stigmata of hysteria, and do not correspond either to root-areas or to the distribution of peripheral nerves. Tender areas are particularly common over hysterical joints and over certain vertebral spines in hysteria, and we frequently notice that the lightest touches cause severe pain, whereas, when the patient's attention is diverted, deep pressure on the same spot may be painless. Pressure on such tender spots may sometimes excite a hysterical attack—"hysterogenic" areas—and in other cases may restrain or stop an attack—"hysterofrenic" areas. To this subject we shall return when discussing the diagnosis of hysteria.

Next in frequency after hysterical hyperæsthesia are the areas of cutaneous hyperæsthesia in tabes. Tactile hyperæsthesia is specially common in the areas where lightning pains are felt, and like these pains it may be one of the earliest evidences of the disease. Thus a patient with incipient tabes, for years before he reached the stage of ataxia, was so hyperæsthetic around the trunk that it was agony for him to pull his shirt on, or to sponge his body when bathing. Tabetic hyperæsthesia may occur not only on the trunk but also on the limbs, and even on the face. Hyperæsthesia is specially frequent around the orbits in cases of tabetic ocular palsies.

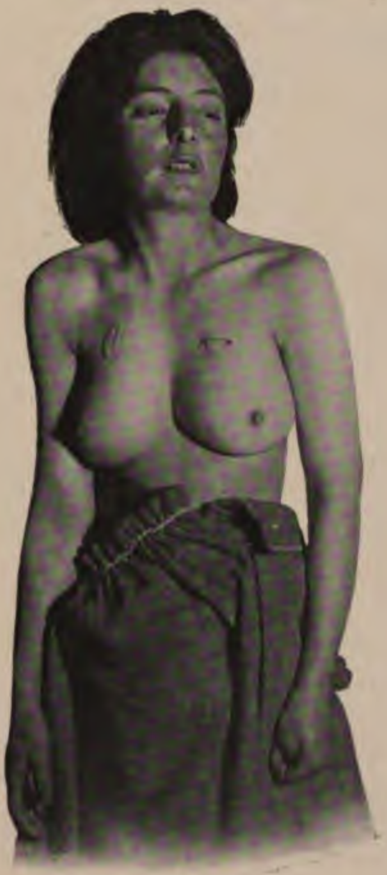

FIG. 68.—Universal anæsthesia in a hysterical patient. Sterilised safety-pins have been pushed through the skin on both sides. Hysterical contracture of left hand is also present.

Hyperæsthesia in the areas of peripheral nerves occurs in the true neuralgias, as in trigeminal neuralgia, where the neuralgic area is often exquisitely tender, especially over the foramina of exit of the various branches. The patient may be unable to wash his face for weeks at a time, since the lightest touches induce a paroxysm of neuralgia. Localised hyperæsthesia sometimes precedes the eruption of herpes zoster, and may persist for weeks or months after the eruption has passed away. Lastly, we may mention the hyperæsthesia of the hands and feet in multiple neuritis, where there is often present a degree of anæsthesia. The coexistence of hyperalgesia to light pressure with anæsthesia to light touches is very characteristic of alcoholic neuritis.

Paræsthesia, or perverted sensation, has much the same diagnostic significance as hyperæsthesia. It may also be mentioned that when a peripheral cutaneous nerve is in process of recovery after an injury, there is often a stage of paræsthesia through which the skin passes before normal sensation is restored.

Anæsthesia.—Universal anæsthesia of the skin and accessible mucous membranes to all forms of stimuli is exceedingly rare, occurring only in hysteria. Fig. 68 shows such a case in a girl, in whom it was possible to push sterilised pins through the skin on both sides of the body without causing pain.

Hemi-anæsthesia always indicates a central affection. In every case of hemi-anæsthesia we must determine whether the disease is functional or organic, and if organic, at what level in the sensory tract the lesion is situated, whether in the cortex, internal capsule, or lower down.

Hysterical hemi-anæsthesia is commoner than organic. It varies in degree, from total anæsthesia down to the slightest degree of comparative blunting of sensation, only discoverable on comparison of the two sides. Not uncommonly it tends towards the "segmental" type, and it is frequently accompanied by other hysterical stigmata, especially by blunting of the special senses on the hemi-anæsthetic side, particularly by concentric contraction of the visual field, and by other features which we shall study later. We should remember that hysteria sometimes coexists with organic disease, thereby complicating the diagnosis.

Organic hemi-anæsthesia may also vary in its degree, from slight to severe anæsthesia; but it is never absolute in degree as in some cases of hysteria. It is generally more marked on the limbs than on the trunk or face, and more intense at the periphery of a limb than at its proximal end. It is never marked off by a sharp line running across the limb, as in the "segmental" anæsthesia of hysteria, but fades gradually in intensity as we pass from the hand to the shoulder. A degree of topo-anæsthesia or topo-analgesia is always present in organic hemi-anæsthesia. The special senses are unaffected (their paths probably do not traverse the internal capsule), with one exception, namely that of vision, in cases where the lesion implicates the optic radiations. But here again we get a homonymous hemianopia, unlike the hysterical contraction of the visual field to which we shall refer later.

If we find that a hemi-anæsthesia is organic in origin, we have then to determine whether the lesion is cortical in situation or whether it is lower, in the internal capsule or elsewhere. In cortical hemi-anæsthesia the other signs of cortical disease will aid the diagnosis. The presence of monoplegia rather than hemiplegia, or the occurrence perhaps of Jacksonian fits, will point to a cortical localisation, remembering that the cortical motor areas are also partly sensory. Cortical anæsthesia is less profound than capsular, and is most distinct at the periphery of the affected limb. Indeed in cortical anæsthesia it is the rule for the trunk to be little or not at all affected. Marked astereognosis and topo-anæsthesia with but slight tactile and motor loss will also point to a cortical lesion, probably in the upper parietal region.

Capsular hemi-anæsthesia, which, by the way, is generally the result of a lesion not actually in the capsule but in the postero-external part of the optic thalamus, is never monoplegic in type but always affects the entire half of the body, including the trunk. The intensity of capsular anæsthesia is deeper than in cortical cases, but not so markedly intensified at the periphery of the limbs. It is also more often associated with hemianopia if the lesion extend backwards, or with hemiplegia, most marked in the leg (but not a monoplegia), if the lesion extend forward towards the pyramidal motor path.

We may also have hemi-anæsthesia from organic lesions of the sensory path below the level of the internal capsule or optic thalamus; in fact, at any level above the sensory (fillet) decussation in the medulla. Such lesions, although rare, can be correctly localised by the coexistence of other signs. Thus a unilateral lesion in the *dorsal aspect of the pons*, implicating the trigeminal nerve or nucleus, together with the remainder of the sensory fibres belonging to the other side of the body, will cause a *crossed hemi-anæsthesia*, *i.e.* anæsthesia of the face on the side of the lesion, and of the arm, trunk, and leg on the opposite side (Fig. 10, p. 14). A lesion of the sensory path in the medulla below the level of the trigeminal nerve, must be more wide-spread laterally to produce a complete hemi-anæsthesia, since the path for temperature and pain is here at some distance from the tactile path (Fig. 9, p. 13).

Anæsthesia also occurs in certain lesions of the spinal cord. We should note, however, that there are many cord diseases in which anæsthesia is absent, such as progressive muscular atrophy, amyotrophic lateral sclerosis and acute anterior poliomyelitis. Disseminated sclerosis is also a disease in which sensory changes are frequently absent. But if the spinal cord be destroyed or divided at a certain transverse level, whether by trauma or by disease such as acute softening, whereby sensory as well as motor paths are interrupted, all the sensory impressions ascending in the posterior and lateral columns (Figs. 7 and 8, pp. 10 and 11) will be lost below the level of the lesion. We then have a *para-anæsthesia*, the upper limit of which corresponds with that of the highest sensory root affected. And since in many of these cases there are irritative or inflammatory processes affecting the roots immediately above the area of destruction, it not unfrequently happens that there is a narrow zone of paræsthesia or of hyperæsthesia immediately above the anæsthetic area. In cases where the cord is gradually compressed by progressive disease in the meninges or vertebræ, there is usually a progressive motor paraplegia with the usual alteration of reflexes. Here anæsthesia appears late in the disease, being preceded by spontaneous subjective sensations or dysæsthesiæ, after which hyperæsthesia appears, and last of all anæsthesia. The anæsthesia of a total transverse lesion implicates all forms of sensation, both superficial and deep.

When a cord lesion is incomplete in its transverse extent, certain forms of sensation may escape. Thus unilateral lesions of the cord produce *Brown-Séquard paralysis*. Most commonly this is the result of a stab or bullet-wound, but it may also be caused by softenings or growths. Or a lesion which was originally more extensive, *e.g.* a hæmorrhage, may clear up so as to become a unilateral one. In the typical Brown-Séquard syndrome, as will be seen from Figs. 8 and 11, there are on the side of the lesion the well-known motor and vaso-motor paralyses, together with loss of sensation in the joints and muscles, whereas on the opposite side there is thermo-anæsthesia, loss of vibration-sense, analgesia and some tactile anæsthesia. If the lesion be above the lumbar enlargement, as is generally the case, the motor paralysis is of the upper neurone type, with spasticity, increased deep reflexes and an extensor type of plantar reflex. If, as sometimes happens in stab-wounds, the lesion destroys the most lateral region of the cord but does not quite reach the middle line, thereby sparing the postero-internal column, the deep structures on the side of the lesion preserve their sensibility. In any case, on the side of the lesion, a narrow zone of anæsthesia exists, corresponding to the posterior root-fibres cut across at the level of the lesion. And above the anæsthesia there is a zone of hyperæsthesia from irritative root-changes.

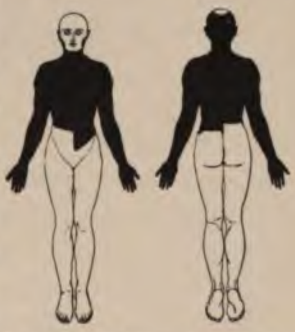
FIG. 69.—Syringomyelia, showing area of thermo-anæsthesia and analgesia in the patient shown in Fig. 70.

Dissociated anæsthesia, often without motor paraplegia, is characteristic of disease in the region of the posterior cornua, as in syringomyelia where there are analgesia and thermo-anæsthesia, with loss of vibration-sense, corresponding to the area of spinal cord affected, whilst tactile sensation remains unimpaired (see Fig. 69). The patient often burns his fingers accidentally without pain, and he may develope painless whitlows—so-called Morvan's disease. He may also have spontaneous joint disintegrations, with fractures and osteophytic or destructive changes in the ends of the bones. In most cases of syringomyelia there also is some atrophy of the anterior cornua; we should therefore be on the look-out for a coexisting muscular atrophy of spinal type, involving especially the small muscles of the hands. If the pyramidal tracts become involved in syringomyelia, a spastic paraplegia is superadded, and sooner or later a degree of scoliosis always developes (Fig. 70).

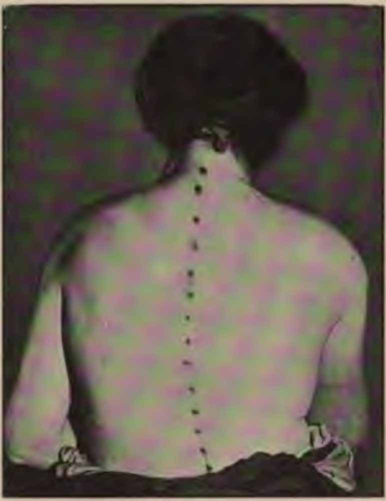

FIG. 70.—Syringomyelia with arthropathy of right shoulder-joint and scoliosis.

But unilateral cord lesions and syringomyelia are not the only diseases which produce dissociated anæsthesia. A small lesion such as an area of softening or of new growth, in the *ponto-cerebellar angle* of the pons at the level of the auditory nerve, will cause deafness of the same side with analgesia and thermo-anæsthesia of the opposite side, tactile sensation being unaffected. If the cerebellar peduncle be involved, there will be cerebellar phenomena also, such as we have already studied.

Tabetic anæsthesia is the commonest of all organic anæsthesiæ. In this disease the sensory loss tends to follow fairly closely the distribution of the posterior roots affected by the tabetic process. Thus it is commoner in the lower limbs than in the upper. Joint-sense and vibration-sense usually become impaired earlier than do cutaneous sensations, whilst analgesia precedes tactile anæsthesia. In the upper limbs the fingers on the ulnar side of the hand are usually affected earlier than the other digits, and there is often a strip of analgesia running longitudinally along the inner side of the whole upper limb, corresponding to the eighth cervical and first thoracic roots (Fig. 71). In many tabetics the ulnar nerve behind the elbow loses its normal sensitiveness on pressure—Biernacki's sign. And on the trunk it is common to find a broad zone of analgesia, and sometimes of tactile anæsthesia as well, the upper border of which is at the level of the second ribs in front. This zone is often incomplete laterally or posteriorly, just as the subjective "cuirass" sensation may be. Analgesia of the glans penis is another early sign of tabes, also loss of the normal tenderness of the testicle on pressure. Tendinous analgesia, on pinching the tendo Achillis, is also present in the majority of tabetic patients. But tabetic anæsthesiæ are not always sharply limited to root areas, and we should seek for confirmatory evidence in the pupils, deep reflexes, cerebro-spinal fluid, &c.

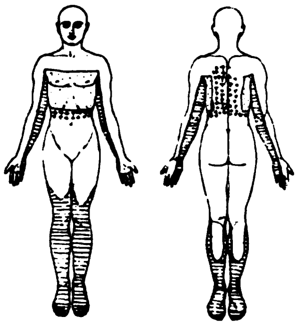

FIG. 71.—Early tabes, showing "cuirass" of subjective girdle-feeling (dotted area) with zone of cutaneous hyperæsthesia below the cuirass, passing up between its limits posteriorly (crosses). Analgesic areas (horizontal shading) in upper limbs (C_8 and D_1 roots) and in lower limbs (L_4, L_5, and S_1 roots).

Anæsthesia in peripheral nerve palsies of sensory or mixed nerve-trunks, is of course confined to the distribution of the affected nerve or nerves. If a mixed nerve-trunk be paralysed, muscular paralysis with atrophy is added to anæsthesia, and the diagnosis, as a rule, presents no difficulties. It must be borne in mind that as a mixed nerve recovers from its paralysis, sensation usually returns before motor power. The anæsthesia following an attack of herpes zoster sometimes lasts for a considerable time after the eruption has disappeared. In the anæsthesia of multiple neuritis, whether resulting from alcohol, diphtheria, diabetes, septic poisoning, or other causes, the disease is generally bilateral and symmetrical, affecting hands or feet or both. Subjective tingling sensations usually precede the anæsthesia, which is of the "glove" and "sock" variety, not marked off sharply as in the "segmental" anæsthesia of hysteria, but shading off gradually at the upper margin (see Figs. 72 and 73). Most cases have also some superadded motor weakness in the form of drop-foot and drop-wrist. Lead paralysis differs from ordinary peripheral neuritis in being entirely a motor palsy, free from sensory changes.

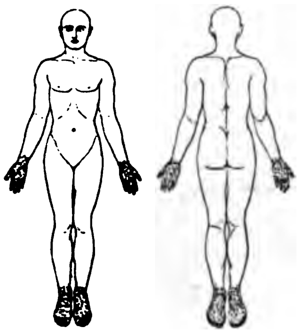

FIG. 72.—From a case of multiple neuritis, showing "glove" and "sock" areas of anæsthesia.

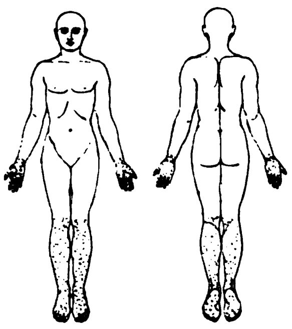

FIG. 73.—From another case of peripheral neuritis, showing "glove" and "stocking" areas of cutaneous paræsthesia.

LECTURE XIII

ORGANIC MOTOR PARALYSIS OF UPPER NEURONE TYPE

In the investigation of the various motor palsies, apart from those affecting the cranial nerves which we have already studied, we should proceed in a definite order.

We commence by inspection of the palsied limb or limbs, noting the posture of the limb, the presence or absence of local muscular atrophy or hypertrophy, the existence of pathological swellings, &c. We then proceed to palpation of the bones and joints, following their outlines and testing their range of passive movement, so as to discover whether the deficiency of active movement may not be due to mechanical causes, such as fractures, dislocations, ankyloses, adhesions or inflammations of bones or joints, and so on. Thus in the case of a semi-comatose lady, whom I saw within a few hours after a carriage accident, there was apparent inability to move the left arm or leg. This might have suggested a lesion of the right cerebrum producing hemiplegia, but a preliminary investigation of the bones showed that the left femur and left clavicle were broken. It was therefore unnecessary to diagnose an intra-cranial lesion of the pyramidal tract, especially as the plantar reflexes were normal on both sides. The result proved the correctness of this view.

We should also observe whether the muscles of the affected part are rigid, stiff and spastic, or whether they are loose, relaxed and flaccid.

Lastly, we proceed to investigate the patient's powers of voluntary movement of the affected limb. And in doing this, it is not enough to tell the patient in general terms, to "move the arm," and so on. We should test each joint and each movement separately, fixing the proximal part of the limb and instructing the patient to perform various movements :—flexion, extension, rotation inwards and outwards, &c., separately and distinctly. Thus, for example, when we direct a patient to pronate his forearm, we must fix the humerus and see that he does not abduct the shoulder to make up for deficiency in pronation. To determine whether a particular muscle is taking part in a movement or not, sometimes requires very close observation, not only by inspection but by palpation of the muscle or of its tendon. But, as Beevor has remarked,[1] the physician "must avoid the error of assuming that the tightening of a tendon from the stretching of a passive muscle is evidence of contraction of the muscle."

Suppose that a particular movement of a limb is deficient, we estimate the deficiency in different ways, according to its degree. If the weakness is slight, we detect it by interposing some resistance, so as to load the muscles. This is accomplished either by means of weights, or, in the case of the hand, by making the patient squeeze our hand or compress an oval spring-dynamometer, or, in the case of the leg, by holding the limb down and making him elevate it against resistance.

If the weakness is more marked, it can be detected without loading the muscles. The weight of the distal part of the limb may already be too much for the muscles to lift, as for example in cases of drop-wrist and drop-foot, due to weakness of the extensors of the wrist and dorsiflexors of the foot. But even in these, a minimal degree of voluntary contraction may perhaps still be present, and can still be detected by placing the limb passively in such a posture that its own weight is no longer a factor, *e.g.* by testing the extensors of the wrist with the forearm midway between pronation and supination, or testing the movements of the elbow by passively abducting the upper arm and getting the patient to flex and extend his elbow in a horizontal plane. Or we may even observe the limb when it is supported on all sides by water, as in a warm bath. In this way we may detect minimal movements. And in such cases we watch carefully, not only for movement of the joint, but for contraction of the tendons of the muscles concerned. These latter may sometimes be felt to contract, when they are too feeble to overcome the inertia of the joint.

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[1] *Croonian Lectures*, 1904, p. 4.

By inspection and palpation we also note whether in the affected limb the muscles are normal in volume and firmness, whether some are enlarged and stronger than usual—*hypertrophy*—or whether some are diminished in size—*atrophy*, so that there is flattening, or even hollowing, in place of the normal muscular contour. In some cases, an apparent increase in volume is accompanied by weakness of the enlarged muscles—so-called *pseudo-hypertrophy*.

If a muscle is atrophied, its electrical reactions, both to faradic shocks and to the continuous galvanic current, should be investigated. The reactions may be normal, or they may be merely quantitatively diminished. Or they may be qualitatively altered, as in the "reactions of degeneration" (see later, p. 346). Or they may be "mixed," where some fibres of the muscle are normal, whilst others intermingled with them are degenerated.

Reaction of a muscle to direct percussion is sometimes of value. For example, in a case where the knee-jerk is lost, if the vastus internus still contracts to direct tapping of its fibres, then the loss of the jerk is not due to absence of muscle-fibres. In certain cases, especially on percussing the pectorals or other flat muscles of the chest, we may observe a wave of contraction dashing outwards suddenly in both directions from the point of percussion, longitudinally along the muscular fibres, and immediately followed by a temporary small muscular swelling at the point of percussion. This phenomenon is called **myoidema**; it is common in pulmonary tuberculosis, but occurs also in many other wasting conditions, not associated with muscular paralysis, and need not detain us further.

The term *paralysis*, when applied to voluntary muscles, signifies loss of the power of voluntary contraction, due to interruption, functional or organic, in any part of the motor path, from the cerebral cortex down to and including the muscle-fibre. This latter part of the definition is necessary so as to exclude such cases as ankylosed joints, where movement is impossible from mechanical reasons without true paralysis. Strictly speaking, paralysis is total loss of voluntary motor power, lesser degrees of impairment being called **paresis**. But we often employ the term paralysis to include partial as well as complete loss of power.

The distribution of motor weakness differs according to the site of the lesion of the motor path. Thus in a unilateral brain lesion, there is usually paralysis of one side of the body, including the face, trunk, and limbs. This is termed *hemiplegia*. A bilateral cerebral lesion produces *diplegia* or *double hemiplegia*, the limbs on both sides of the body being affected. Paralysis of a single limb resulting from a cerebral lesion is termed cerebral *monoplegia*. Spinal or peripheral monoplegia is rare.

Paralysis of the limbs resulting from a lesion of the spinal cord is most commonly bilateral—*paraplegia*—and usually affects the legs alone; but if the lesion be in the cervical region, it affects both the arms and legs. It must be distinguished from a cerebral diplegia. In rare cases both arms may be paralysed from a spinal lesion, with little or no affection of the legs; this is *brachial paraplegia*. A unilateral spinal lesion may also cause a monoplegia, but this is uncommon. The term *crossed* or *alternate hemiplegia* means that as a result of a single lesion there is paralysis of some parts on the right side and of others on the left. For example, a lesion in the right side of the pons at the level of the facial nerve will cause paralysis of the right side of the face and of the left arm and leg. There are, of course, other varieties of crossed paralysis.

When paralysis is due to a lesion of a peripheral nerve, it may either be asymmetrical, when the motor weakness is limited to one or more nerve-trunks, as in most traumatic nerve-palsies, or more commonly it is bilateral and symmetrical, as in the various forms of toxic neuritis, affecting either the upper or lower or all four limbs.

If paralysis be due to primary affection of the muscles themselves, as in the myopathies, its distribution is usually bilateral, and it affects all four limbs and sometimes even the face.

We must remember that it is not uncommon to meet with multiple lesions in a single case; yet, in diagnosis, it should always be our endeavour to try to account for all the symptoms by a single lesion.

Suppose, then, that a patient is suffering from motor paralysis, (mechanical impediments having been excluded), the first question is —Is the paralysis *functional or organic?* If it is organic, we proceed to the further questions—*Where* is the lesion situated? (anatomical diagnosis), and *what* is its nature? (pathological diagnosis).

Is the Paralysis Functional or Organic?—Sometimes the distinction between functional and organic motor paralysis is easy; at other times it is a matter of considerable difficulty, cases of early disseminated sclerosis being particularly liable to be mistaken for hysteria; moreover, it is possible to have a combination of functional and organic disease in the same patient.

More detailed consideration of the diagnostic features of hysteria will be postponed till a later lecture (see p. 307), and we shall only here refer to some of the main features which enable us to decide that a case is organic rather than functional. Firstly, the history of the case often guides us; for instance, functional paralysis frequently follows an emotional shock or a prolonged mental strain, whereas traumatism, as in railway accidents, is equally liable to cause functional or organic disease.

There are two classes of signs and symptoms which point to functional rather than to organic disease—firstly, the absence of characteristic signs of organic disease, and secondly, the presence of certain phenomena peculiar to functional disease. Muscular atrophy, whilst much less frequent in functional than in organic palsies, is not pathognomonic of functional disease. Thus Fig. 179 (p. 326) shows a case of hysterical monoplegia with extensive muscular atrophy, a rare combination. But the electrical reactions of degeneration never occur in functional paralysis. Their presence signifies undoubted organic disease, somewhere in the spino-muscular neurone. Paralysis of a single muscle is pathognomonic of organic disease; it never occurs in functional paralysis, which affects whole muscle-groups or, to speak more accurately, whole movements. On the whole then, the diagnosis between functional and organic paralysis is easy if the organic palsy is of the lower motor neurone type. It is chiefly when the organic lesion is in the upper or cortico-spinal neurone that difficulty is liable to occur, *i.e.* in cases where there is little or no muscular atrophy, and where the electrical reactions are normal. In such cases the presence or absence of other hysterical stigmata is of great value.

A valuable sign of organic as contrasted with hysterical hemiplegia is Babinski's "combined flexion of the hip and trunk," a phenomenon almost invariably present in organic cases. To elicit this the patient lies flat on his back on a smooth hard surface, such as a table, with his arms crossed in front of his chest. We then ask him to sit up without using his arms. As he does so, the organically paralysed lower limb becomes flexed at the hip and the heel raised from the table. Meanwhile the shoulder on the healthy side is carried forwards, as if to counterpoise the contralateral lower limb. In hysterical hemiplegia this sign is absent, and the hysterically paralysed limb remains unraised. Another useful test between organic and functional paralysis is the phenomenon of Grasset and Gaussel,[1] which is also confined to organic cases. This consists in inability on the part of the organically hemiplegic patient to raise both lower limbs simultaneously from the surface when lying down as before, whilst he is still able to lift either lower limb separately. The reason for this peculiarity is that in organic hemiplegia the patient, when he tries to lift both lower limbs at once, is unable to fix the pelvis. In testing for this sign, we must be careful to see that the two legs do not touch each other, since the patient often tends involuntarily to help up the paralysed limb by means of the sound one. Of course, the sign is only present in cases of incomplete hemiplegia. Another way of showing the same phenomenon is to direct the patient to raise the lower limb of the paralysed side and hold it in the air. If we now grasp the sound leg and raise it up, the other limb at once falls down again, because the pelvis cannot be steadied by the muscles on the paralysed side. On the other hand, if the patient first raises the sound leg and we then passively lift the paralysed one, the sound limb still remains in the air, the pelvis remaining fixed by the non-paralysed muscles of the healthy side. In hysteria there is no such difference between the separate and the simultaneous raising of the legs.

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[1] *Revue Neurologique*, 1905, p. 881.

In some cases of spastic paraplegia the rigidity of the lower limbs is of diagnostic value. Thus when we passively lift one lower limb off the bed and find that the other lower limb is thereby lifted up as well, we may be practically certain that the rigidity and paralysis are organic and not functional.

A careful study of the reflexes is also of the utmost importance. The presence of an extensor plantar reflex in a patient beyond the age of infancy is pathognomonic of organic disease (see later, p. 271). And the deep reflexes, whilst they may be exaggerated both in functional and in organic paralysis, are usually normal in functional cases. True ankle-clonus of organic disease is generally readily distinguished from the "pseudo-clonus" of functional disease. Absence of the deep reflexes may occur in organic, never in functional disease.

Incontinence of the bladder and rectum is not uncommon in organic diseases of the spinal cord and brain, but does not occur in functional paraplegia.

Where is the Organic Lesion?—Suppose we have come to the conclusion that the patient's motor paralysis is organic in type, we have to ask ourselves at what point in the motor path the lesion is situated. First, we must decide whether the lesion is in the upper (cortico-spinal), or in the lower (nucleo-muscular) motor neurone. The distinctive characters of these two types are as follows:—

ORGANIC MOTOR PARALYSIS

Upper (cortico-spinal) Neurone. Supra-Nuclear Paralysis.	**Lower (spino-muscular) Neurone. Nuclear and Infra-Nuclear Paralysis.**
1. Diffuse muscle-groups affected, never individual muscles.	1. Individual muscles may be affected.
2. Spasticity of paralysed muscles.	2. Flaccidity of paralysed muscles.
3. No muscular atrophy, except from disuse.	3. Atrophy of paralysed muscles.
4. Electrical reactions normal.	4. Reactions of degeneration.
5. Deep reflexes in paralysed limbs present, and usually increased.	5. Deep reflexes of paralysed muscles diminished, and often absent.
6. If foot affected, plantar reflex extensor in type.	6. Plantar reflex, if present, is of normal flexor type, (unless flexors of toes are themselves paralysed.)

Let us consider some of these points more in detail. Paralysis due to an upper neurone lesion never affects an individual muscle, but always a diffuse muscular group. The converse, however, is not true, and we must remember that even a lower neurone lesion may produce a diffuse paralysis, where a series of adjacent nerves or nuclei are affected. But if individual muscles are picked out by paralysis, the adjoining or intermingled muscles being perfectly normal, the cause is certainly a nuclear or infra-nuclear lesion.

Spasticity of the paralysed muscles in supra-nuclear lesions does not set in immediately after the onset of a sudden lesion, but usually developes gradually in the course of from one to three months. So that in a typical supra-nuclear lesion, as for example in apoplexy, there is an initial period of flaccidity, gradually replaced by the so-called "late rigidity." The degree of this spasticity varies in different cases. We estimate it by moving the patient's joints passively, and comparing their resistance with that of a healthy limb.

In an upper neurone lesion, the muscles of the paralysed limb, in the vast majority of cases, undergo no appreciable atrophy, save perhaps to a very slight degree from disuse. But there are occasional exceptions to this rule, as in some cases of hemiplegia associated with muscular atrophy, especially in the region of the shoulder or in the intrinsic muscles of the hand. Such atrophies are often (but not always) secondary to arthritic changes in the joints. But however intense the amyotrophy of hemiplegia may be, the electrical reactions of degeneration are never present. Degenerative reactions, (commonly referred to as "R.D.,") are pathognomonic of a nuclear or infra-nuclear lesion. Not that R.D. are necessarily present in every lower neurone lesion, for a slight lesion of a nerve-trunk may produce muscular palsy without R.D., and in many nuclear lesions, for example in progressive muscular atrophy, the reactions in the affected muscles are mixed, owing to the fact that degenerated and healthy muscle-fibres are intermingled in the same muscle, the former giving R.D., the latter being normal in reaction. Again, in the motor weakness occurring in the different varieties of myopathy, there is simple diminution both to faradism and galvanism, but no true R.D., even in the most advanced cases. The reflexes in upper and lower neurone lesions will be dealt with more fully in a later lecture (p. 268).

With reference to the differential diagnosis between an upper and a lower neurone lesion, it will be observed that no single sign of the six we have mentioned is pathognomonic, yet the sum of the various points usually enables us without difficulty to settle with which of the two neurones we have to deal. Sometimes there is a combined lesion of upper and lower neurones, as in a transverse myelitis or a myelomalacia. Here the phenomena at the level of the lesion will be of a flaccid, lower neurone type, due to destruction of the anterior cornua and anterior roots, whilst below that level there is a spastic paraplegia of cortico-spinal type, from interruption of the pyramidal tracts.

Upper Neurone Type of Motor Palsies.—The signs and symptoms vary according to the level at which the cortico-spinal tract is damaged. The following are the chief sites at which a lesion may occur, and the diagnostic signs of each (see Fig. 5, p. 7).

A cortical lesion in the pre-central convolution, is often localised to a single limb and is more likely to produce a monoplegia than a hemiplegia, since only a very extensive cortical lesion would produce a complete hemiplegia, affecting face, arm and leg. What we usually find is either a pure monoplegia—crural, brachial, or facial, or, if the lesion be somewhat larger, an associated monoplegia—brachio-crural, or facio-brachial. Cortical motor paralysis is often associated with local epileptiform attacks of the paralysed limb, because disease may irritate the cortex in addition to paralysing it. The monoplegic limb often shows a cortical type of anæsthesia, which, as we have already seen, is slight in degree, more marked at the periphery of the limb, and often transient in duration.

A strictly localised subcortical lesion is often indistinguishable from a cortical one, save by the absence of irritative epileptiform phenomena; and in many cases the lesion is both cortical and subcortical.

A lesion in the motor path at the level of the internal capsule, inasmuch as all the pyramidal fibres have by this time converged to form a compact strand, produces no longer a monoplegia but a complete hemiplegia, affecting face, arm and leg. There are no Jacksonian convulsions as in a cortical lesion. If the capsular lesion extends backwards from the motor into the sensory tract, or into the optic thalamus, there may be a coexistent hemi-anæsthesia, but this is not common. A thalamic lesion is sometimes associated with hemi-athetosis of the hemiplegic side; this athetosis does not appear immediately after an attack of apoplexy, but developes gradually in the course of many weeks. A still more extensive lesion, extending backwards along the capsule from the motor tract, through the sensory path and into the optic radiations, will cause hemiplegia, hemi-anæsthesia and hemianopia.

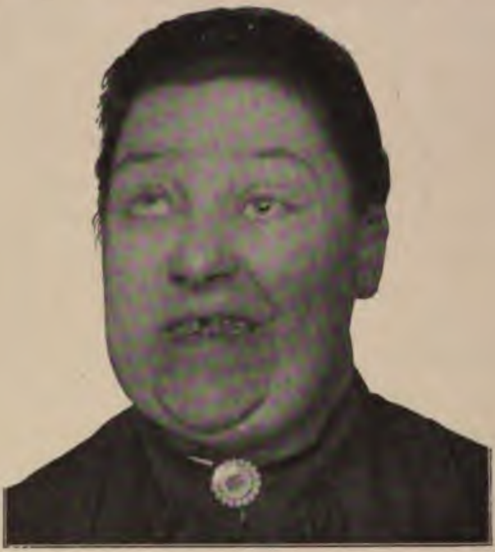

FIG. 74.—Lesion of left crus cerebri—"Weber's syndrome." Patient is looking upwards and attempting to show the teeth on both sides. There is dilatation of the left pupil and paralysis of the left superior rectus, together with hemiplegia of the left face, arm and leg.

A lesion in the crus cerebri is recognised by the coexistence of third nerve palsy on one side, with hemiplegia of the opposite face, arm and leg, usually most marked in the face. This variety of alternate paralysis is known as *Weber's syndrome* (see Fig. 74). The third nerve palsy is often incomplete. When the lesion extends into the tegmentum and implicates the neighbourhood of the red nucleus, it may produce a unilateral tremor or a hemiataxy of the hemiplegic side, combined, as before, with a third nerve affection on the side of the brain lesion; this combination is known as *Benedikt's syndrome*. If the lesion extends outwards so as to implicate the optic tract as it winds round the outer side of the crus, there may be superadded a hemianopia.

As we come downwards along the pyramidal tract into the pons and medulla, the type of hemiplegia changes; there is no longer third nerve palsy, but on reaching the level of the facial nerve, another variety of alternate hemiplegia appears. This consists of facial palsy, peripheral in type, on the side of the lesion, together with hemiplegia of the arm and leg on the opposite side, the so-called *Millard-Gubler syndrome*. Other cranial nerves on the side of the pontine or bulbar lesion, for example, the trigeminal, the sixth, or the hypoglossal, may be affected on the side of the pyramidal lesion, and, as in the Millard-Gubler syndrome, may co-exist with hemiplegia of the opposite arm and leg, but such cases are rare. As they descend through the medulla the pyramidal tracts of opposite sides converge and eventually lie so close together that at this level a strictly unilateral lesion seldom occurs, there being usually damage to both pyramidal tracts affecting the limbs of both sides, though perhaps in unequal degree. And together with this, there are "bulbar" symptoms —disorders of articulation, phonation, or deglutition, from implication of the tenth, eleventh, and twelfth cranial nerves or nuclei.

The diagnosis of motor paralysis due to lesions of the pyramidal tract within the cord depends on the level of the lesion. The two pyramidal tracts decussate at the lower end of the medulla oblongata, so that a unilateral lesion of the spinal cord produces a homolateral instead of a contralateral motor paralysis. If the lesion be in the cervical region, the arm and leg on the corresponding side will be affected; but if it be situated below the cervical enlargement, the leg on the side of the lesion suffers alone. A primary unilateral lesion of the cord generally interrupts not only motor but sensory paths, and produces the well-known Brown-Séquard paralysis, to which reference has already been made.

Bilateral motor paralysis of upper neurone type is due to bilateral lesions, which may be situated either in the brain or in the spinal cord. When both pyramidal tracts are affected within the brain (and the commonest cause is a double focus of softening, in the posterior part of the lenticular nucleus, though less commonly the lesions are cortical or subcortical) a double hemiplegia is the result. In these cases of double-hemiplegia or diplegia, there are, besides the signs of hemiplegia on both sides (frequently unequal in degree), what are known as "pseudo-bulbar" phenomena. In pseudo-bulbar paralysis, the symptoms of which we have already studied (p. 93), it is uncommon for the two attacks of hemiplegia to occur simultaneously on the two sides; they more usually occur successively, and it is only after the hemiplegia has become bilateral that the pseudo-bulbar symptoms appear. Such patients are generally excessively emotional, tending on slight provocation to laugh or, more frequently, to weep with a peculiar "spastic" wail, and an unnatural slowness of expressional movement.

Bilateral pyramidal lesions within the spinal cord produce paraplegia, affecting all four limbs if the lesion be above the cervical enlargement, but affecting the lower limbs alone if the lesion be below the cervical region; it is commonly of the ordinary spastic type, with increased deep reflexes. And if the sensory tracts be interrupted by the same lesion as that which has affected the motor tracts, we have superadded an anæsthesia, the upper limit of which corresponds to that of the highest affected segment. Such cases of combined sensory and motor paralysis usually have loss of control of the sphincters. If the cord lesion be sufficiently extensive to implicate the anterior cornua, there will be muscular atrophy, localised to the segment affected, *i.e.* at the upper boundary of the spastic paraplegia. But it is important to remember that if the lesion of the cord be one which completely divides it (*e.g.* a stab or bullet-wound), so that there is no connection between the cord-segments above and below the lesion, the paraplegia is then flaccid in type and the deep reflexes are absent in the paralysed limbs. The plantar reflexes, however, persist and are of the extensor type, be the lesion complete or incomplete.

Sometimes the lesion is not horizontal, but higher on one side than the other, and then the upper limit of the anæsthesia will be correspondingly uneven on the two sides, and the distribution of muscular atrophy from anterior cornual destruction correspondingly asymmetrical.

We also meet with cases of bilateral spastic paraplegia without any affection of sensation. Such cases may be examples of slowly-progressive primary lateral sclerosis, a rare disease, or what is more usual, of amyotrophic lateral sclerosis, where the signs of a progressive muscular atrophy are superadded to rigidity of the lower limbs with increased deep reflexes. A pure motor paraplegia is more frequently due to disseminated sclerosis, to an imperfectly recovered transverse myelitis, or to some other vascular lesion, as thrombosis or hæmorrhage, in which the sensory functions have subsequently become restored, the motor tracts remaining permanently sclerosed. The history of the case is sufficient to distinguish these diseases.

Syringomyelia, where it affects the pyramidal tracts, may also produce a spastic type of paraplegia; but it is readily recognised by the accompanying characteristic dissociated anæsthesia, to which we have already referred (p. 169), and frequently by the coexistence of atrophic changes in the bones, joints, and muscles, muscular atrophy occurring when the anterior cornua are implicated in the gliomatous process.

LECTURE XIV

ORGANIC MOTOR PARALYSIS OF LOWER NEURONE TYPE

Motor Palsies of Lower Neurone Type.—Here, as in upper neurone lesions, the signs and symptoms differ according to the level at which the spino-muscular neurone is diseased. The most important diagnostic fact, for localising purposes, is the presence or absence of sensory phenomena. If in a lower neurone motor palsy sensory changes are present, we have to do with a lesion of a mixed nerve, that is, of a nerve containing sensory as well as motor fibres. If, on the other hand, sensory changes are absent throughout the course of the disease, the spino-muscular neurone is probably affected, either before it is joined by the sensory fibres (*i.e.* the lesion is in the anterior cornu or anterior nerve-root), or after it has parted company with them (*i.e.* the lesion is in a purely motor nerve-branch or in the muscle itself).

A lesion of the *anterior cornu* within the cord (as of its homologue in the motor nuclei of the bulb) is unassociated with any sensory paralysis, and therefore produces a pure motor palsy of the corresponding muscle fibres. A lesion of the *anterior nerve-root*, emerging from the anterior cornu, produces identical signs, and is often indistinguishable from an intra-spinal nuclear lesion. In nuclear or anterior-root lesions, therefore, we find pure motor palsy, of lower neurone type, unassociated with any sensory change. The commonest examples of such lesions are acute polio-myelitis (infantile paralysis of spinal type), chronic anterior polio-myelitis (progressive muscular atrophy), and certain types of lead paralysis. Landry's paralysis is a pure motor paralysis of the whole spino-muscular neurone, to which we shall refer presently. A nuclear or anterior-root lesion is further characterised by the "root" distribution of the motor paralysis, so that in this respect it differs from the paralysis due to a lesion of a peripheral nerve (see Tables of Root Distribution, p. 27). Lesions of peripheral mixed nerves are always associated, at the onset at least, with sensory changes. In the case of lesions of peripheral purely motor nerves (*e.g.* the nerve of Bell to the serratus magnus), the distribution of the motor palsy is totally unlike that of a nuclear or anterior-root lesion.

To distinguish between a nuclear and an anterior root lesion is sometimes difficult, and may in some instances be impossible. The coexistence of spastic phenomena corresponding to lower parts of the cord points to an intra-spinal lesion, and indicates a co-existing lesion of the adjacent pyramidal tract. Total escape of the pyramidal tract, on the other hand, would suggest an anterior root lesion, though not necessarily so, since acute anterior polio-myelitis does not affect the pyramidal tract. Another point which may sometimes help us is the subsequent course of the disease;

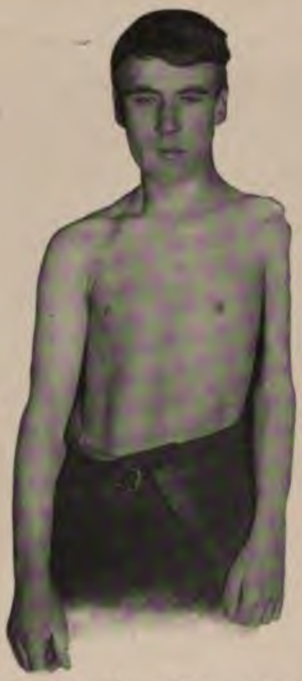

FIG. 75.—Old acute anterior poliomyelitis, with atrophy of deltoid and biceps, and deficient growth of the humerus on the left side.

if the paralysed muscles recover, this is in favour of an extra-medullary anterior root lesion rather than an affection of the anterior cornu, inasmuch as regeneration of nerve-fibres only occurs in extra-spinal lesions, an intra-spinal lesion of the grey matter being irreparable.

A pure anterior cornual lesion, with its absence of cutaneous anæsthesia, can only be confounded with a lesion either in a purely motor nerve, or with one in the muscle-fibres themselves. The history of the onset of the disease is of great importance, so also is the exact distribution of the muscular paralysis. The two chief diseases limited to the anterior cornua are infantile paralysis and progressive muscular atrophy. *Acute anterior polio-myelitis* or infantile spinal paralysis has a sudden onset, generally febrile. Quite a large number of muscles, sometimes in all four limbs, may be paralysed at the beginning. But in a week or so most of them clear up, leaving a residuum, almost always unilateral, of paralysed muscles which undergo rapid wasting. There is no exaggeration of tendon reflexes below the level of the lesion, since the pyramidal tract is unaffected. Figs. 75, 76, and 77 are examples of cases of old infantile paralysis, showing the extreme degree of

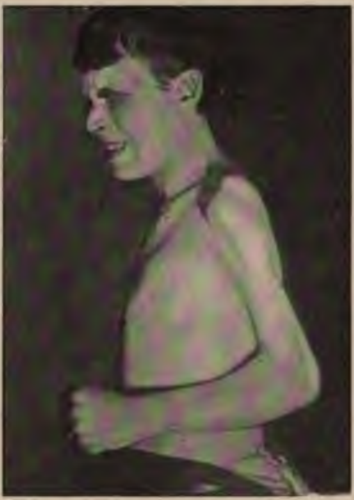

FIG. 76.—Old infantile paralysis with total loss of deltoid and biceps. Shows action of the supinator longus in flexion of elbow.

atrophy which results, and if the disease occurs in childhood, as is most often the case, the subsequent want of growth in the limb.

Chronic anterior polio-myelitis, or progressive muscular atrophy, has a gradual, insidious onset. It occurs almost always in adults and, though beginning unilaterally, generally becomes bilateral. It usually shows itself first in the small intrinsic muscles of the hands (Fig. 78); more rarely it begins in the shoulder muscles. In this disease we observe fibrillary worm-like tremors in the wasting muscles, whose electrical reactions are a mixture of R.D. with healthy reactions. This is because here and there in the diseased area a healthy anterior cornual cell survives, together with its corresponding healthy muscle-fibre. If the adjacent pyramidal tracts be sclerosed, we have amyotrophic lateral sclerosis, in which the deep reflexes are exaggerated and the plantar reflexes extensor in type.

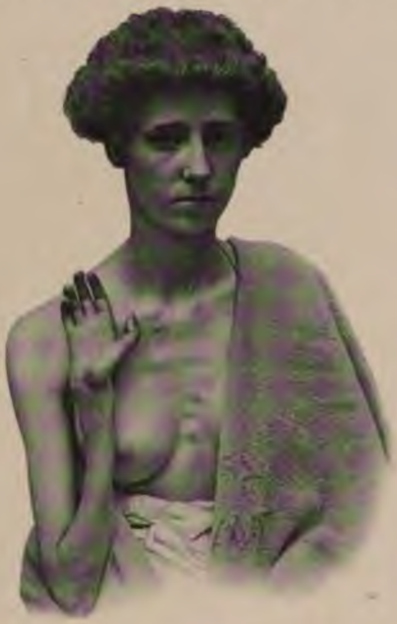

FIG. 77.—Old infantile paralysis (acute anterior poliomyelitis). Paralysis and atrophy of all the muscles below the elbow, with exception of supinator longus.

There is a peculiar form of muscular atrophy which is hereditary and runs in families, known from its distribution as Tooth's "*peroneal*" type, or as the *progressive neuritic amytrophy* of Charcot and Marie. It comes on in childhood, attacking the peripheral parts of the limbs, more often the lower limbs and the peroneal muscles, and gradually producing weakness with contractures. Talipes equino-varus appears, for which tenotomy is often done, as was the case in the patient shown in Figs. 79 and 80. Later, the intrinsic muscles of the hands undergo wasting (Fig. 81). In fact, early claw-foot and claw-hand in young people are almost pathognomonic. The disease hardly ever extends to the muscles of the hips or shoulders. The facial and trunk muscles also escape. It is interesting to note that when all the muscles below the knees are paralysed, the patient may still be able to walk alone, though the gait is high-stepping from drop-foot. This was so in the patient here figured. The deep reflexes are lost in the atrophied

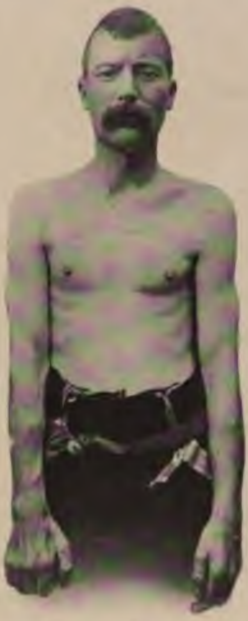

FIG. 78.—Progressive muscular atrophy in a man aged 32. The patient was also tabetic.

muscles. Thus in this patient the ankle-jerks were lost, whilst the knee-jerks remained brisk since the thigh muscles were unaffected. Pathologically the disease is associated with atrophy of the anterior cornual cells, whilst the anterior nerve-roots are healthy. But there is marked degeneration in the peripheral nerves supplying the affected muscles. And there is also a curious degeneration in the posterior columns, closely resembling that of tabes dorsalis.

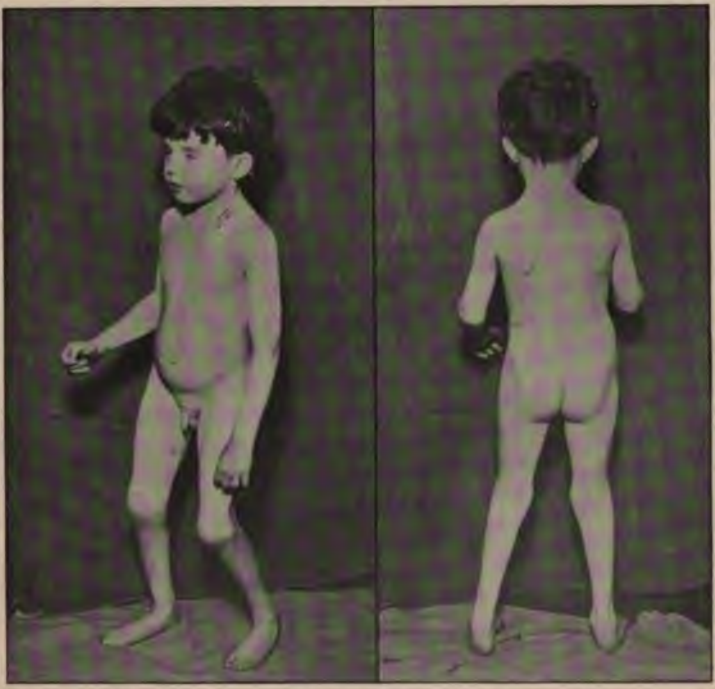

FIG. 79. FIG. 80.

Peroneal type of muscular atrophy (Charcot—Marie—Tooth). In spite of total paralysis below the knees, the patient was still able to stand and walk.

A lesion of a *spinal nerve* after the union of its anterior with its posterior root, but above the point where it divides into branches to form plexuses or individual nerves, is characterised by a combination of motor and sensory paralysis, the distribution of which is not according to peripheral nerves, but according to root areas, motor and sensory (see Tables of Muscular Localisation, p. 27, also Fig. 16, p. 28). Thus, for example, Fig. 82 is the photograph of a sailor who received a violent blow on the right side of his neck from an iron winch. This produced paralysis of the deltoid, supra- and infra-spinatus, biceps, brachialis anticus, and supinatores longus and brevis, together with an area of cutaneous anæsthesia along the outer side of the whole upper limb, from the shoulder to the hand. All this would be difficult of explanation on the theory of multiple injuries to the numerous peripheral nerves which supply these various parts. But the motor distribution is that of the fifth cervical root, whilst the anæsthesia of the hand corresponds to the fifth and a small part of the sixth root, and as a matter of fact this lesion was subsequently verified by operation.

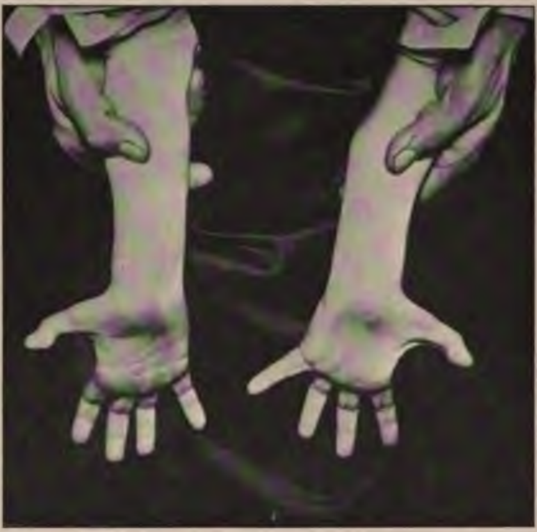

FIG. 81.—Peroneal type of muscular atrophy (Charcot—Marie—Tooth). Showing atrophy of intrinsic muscles of hands.

Lesions of *peripheral mixed nerves*, when complete, are usually easy of diagnosis, inasmuch as there are paralysis and atrophy of all the muscles supplied by the particular nerve, together with anæsthesia in the area of its cutaneous distribution. It is unnecessary to discuss the signs of paralysis of all the various mixed nerves. The question is that of anatomy. Let us select one or two illustrative examples. Ulnar paralysis affects certain muscles of the hand and forearm, of which the most important diagnostically are the interossei and the two ulnar lumbricales. Owing to paralysis of the interossei, extension of the interphalangeal joints is impaired, especially in the two ulnar fingers whose lumbricales are also affected. Anæsthesia of one and a half fingers and of the corresponding part of the hand is also present (see Figs. 83 and 84, which are taken from a case where

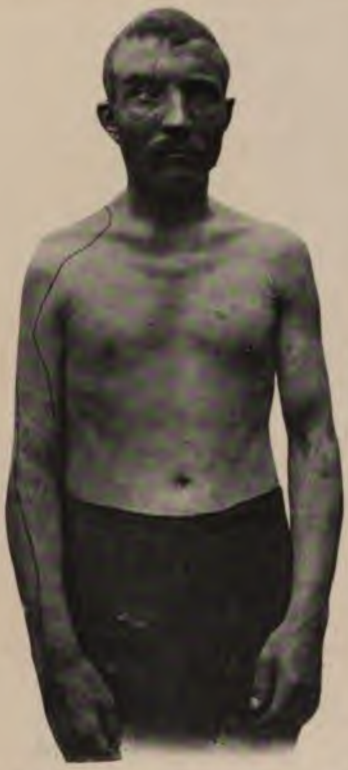

FIG. 82.—Case of rupture of C_5 root in a sailor aged 29. There are atrophy and paralysis of deltoid, supra- and infra-spinatus, biceps, brachialis anticus, and supinatores longus and brevis, together with anæsthesia along the outer side of the limb, from the neck to the thumb and index (in the area indicated by black line). The figure shows the atrophy of the deltoid with downward displacement of the limb at the shoulder-joint.

the nerve was cut by the bursting of a soda-water bottle). In old-standing cases, the unopposed common extensor of the fingers undergoes contracture and produces a claw-hand, this for the same reason being most evident in the two ulnar fingers. The hypothenar eminence also becomes flattened and the palm hollowed, so that the flexor tendons become visible beneath the skin.

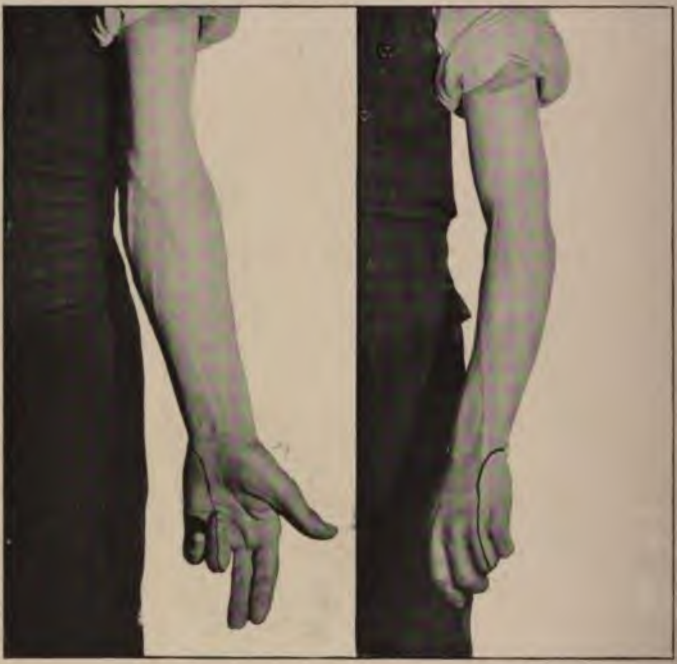

FIG. 83. FIG. 84.

Figs. 83 and 84.—Ulnar paralysis, from a wound of the nerve behind the internal condyle of the humerus. The area within the black line is anæsthetic.

Figs. 85 and 86 are from a case of division of the sciatic nerve in the thigh in a young soldier from a bullet-wound. It shows how all the muscles below the knee are atrophied and paralysed, the hamstring muscles having escaped, since the nerve was divided below the level of the hamstring branches. Besides the muscular atrophy with the usual drop-foot, we have anæsthesia in the areas of the peroneal, musculo-cutaneous, anterior tibial, short saphenous and both plantar nerves.

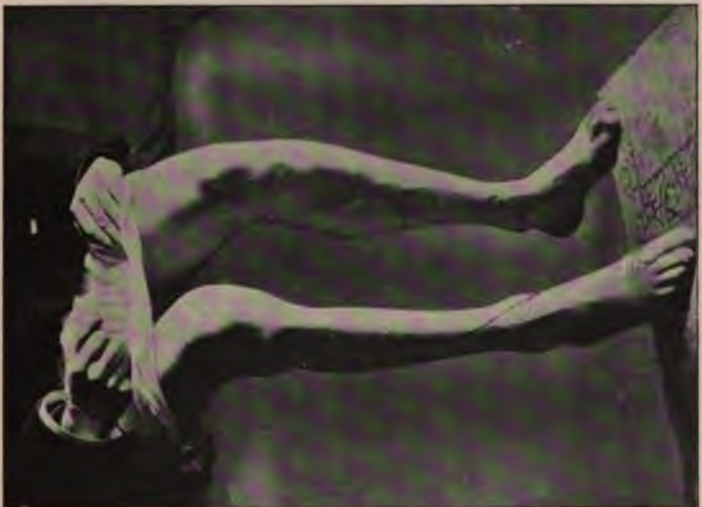

FIG. 85.

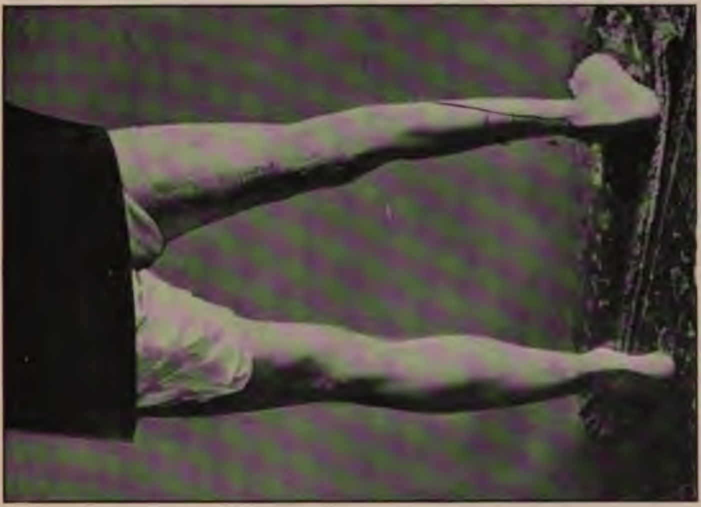

FIG. 86.

Figs. 85 and 86.—Paralysis of great sciatic nerve on right side, from bullet wound of thigh. Showing muscular atrophy below knee, with drop-foot. The black line indicates the upper limit of the anæsthesia.

As a mixed nerve recovers, sensation returns more rapidly than motor power, and we must therefore be prepared to find that in a long-standing case the sensory loss is less complete than in a recent one, or it may occur that sensation is quite restored when motor power has not yet returned. This rule, however, is by no means invariable.

As an example of paralysis of a pure motor nerve, we may select the posterior thoracic, or nerve of Bell, which supplies the serratus magnus. Fig. 87 is from such a case, and it shows the characteristic "winging" of the scapula when the patient holds his arms horizontally forwards.

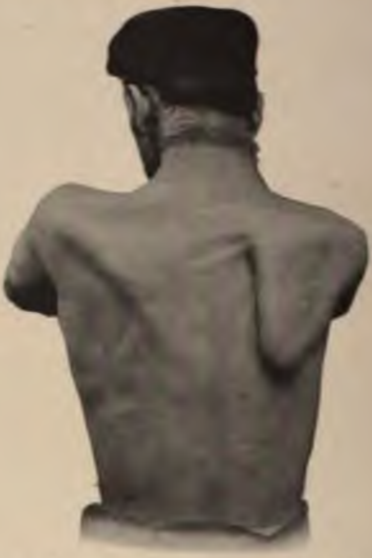

FIG. 87.—Paralysis of serratus magnus on right side. The patient is holding both arms horizontally forwards. The lower fibres of the trapezius are also paralysed.

When a pure motor paralysis of lower motor neurone type recovers, this indicates that it must have been of extra-medullary origin, since regeneration of nerve-fibres does not occur within the central nervous system.

Besides such lesions of individual nerve-trunks, we have also to bear in mind the so-called *multiple* or *peripheral neuritis*—a very common disease, affecting the mixed nerves symmetrically on both sides, sometimes in the arms, sometimes in the legs, sometimes in all four limbs, and even also other nerves such as those of the soft palate, diaphragm, and so on. Such neuritis, when affecting mixed nerves, is easy of diagnosis. The distribution of the sensory and motor abnormalities is characteristic. The patient has subjective tingling feelings in the hands and feet, and on examination we find diminution of sensation to light touches in the "stocking" and "glove" areas of the limbs, often with extreme hyperæsthesia of the soles to pressure. Moreover, the muscles of the limbs are exquisitely tender on being grasped. The motor paralysis, of the lower motor neurone type, specially affects the anterior tibial and peroneal groups in the legs, producing drop-foot, and the extensors of the wrists and fingers, producing drop-wrist (Fig. 88).

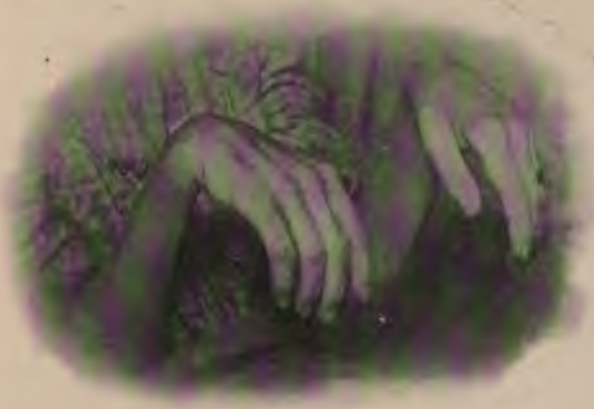

FIG. 88.—Alcoholic neuritis with drop-wrist.

The commonest causes of multiple neuritis are chronic poisoning by alcohol, arsenic (specially associated with cutaneous eruptions), the post-diphtheritic poison, beri-beri (associated with œdema of the limbs and of certain serous cavities), diabetes, septicæmia, and other poisons. *Lead neuritis* is a peculiar form which practically never attacks the sensory fibres. The upper limbs are generally affected, the muscles attacked being the extensors of the fingers and wrists, producing wrist-drop. The long extensors of the thumb become paralysed later, the extensor ossis metacarpi pollicis remaining unaffected. The supinator longus escapes, so that the disease contrasts with musculo-spiral paralysis, with which it might be confused on superficial examination. The blue line on the gums and other signs of plumbism also aid us in the diagnosis.

Sometimes the lower motor neurones are paralysed in their entire extent, from anterior cornu to periphery, the disease commencing in the lowest spinal roots and spreading upwards towards the bulbar motor neurones. This affection, known as *acute ascending paralysis*, or *Landry's paralysis*, might be confounded with an ordinary peripheral neuritis were it not for the absence of sensory changes. The paralysis, commencing in the legs, and spreading up the trunk to the arms, is of the usual flaccid type with loss of reflexes. There is no time for muscular atrophy or reactions of degeneration to develope, and if the respiratory muscles become paralysed the patient dies, generally within a week from the onset. The sphincters remain unaffected. Various organisms have been cultivated from the peri-dural tissues in such cases. The spleen is frequently enlarged, as in certain other infective disorders.

Before leaving the subject of nerve lesions of lower motor neurone type, we must refer to lesions of the *cauda equina*, the mass of nerve-roots, lumbar, sacral and coccygeal, contained within the lowest part of the spinal theca. According to the roots affected, anterior or posterior, we have motor and sensory symptoms, both distributed in "root" fashion, not according to peripheral nerves. The clinical picture varies according to the level of the lesion. When the whole cauda is involved, we find paralysis (of lower motor neurone type) of all the muscles of the lower limbs, together with anæsthesia below the folds of the groins in front, including the genitals, and below the upper part of the buttocks behind, together with loss of control of the bladder and rectum. If the first, second, and third lumbar roots escape, the anæsthesia is less extensive (Fig. 16, p. 28), sparing the upper part of the thighs. If the third lumbar roots escape, thereby sparing the quadriceps, the motor paralysis is correspondingly less, and the knee-jerks survive, though there is still paralysis of the glutei and hamstrings and of all the muscles below the knees, with loss of ankle-jerks and paralysis of bladder and rectum as before.

If the lesion be lower still, the area of paralysis, motor and sensory, is correspondingly diminished. Below the second sacral roots there is no paralysis of the lower limbs, but there is a characteristic " saddle-shaped " area of anæsthesia on the buttocks, perineum, scrotum and penis, with a small strip running from the perineum down the postero-internal aspect of the thighs. Here also the bladder and rectum are uncontrolled, and the anal reflexes are lost, but there is no motor weakness of the lower limbs, and all the reflexes of the lower limbs are normal. And so on, the area of paralysis diminishing as we descend, until when there is a lesion of the fourth and fifth sacral and the coccygeal roots, the only signs are paralysis of the levator ani, with anæsthesia of the anus and perineum, the sphincter ani remaining intact.

The chief difficulty in the diagnosis of lesions in this region is to distinguish between a lesion of the cauda and one of the *conus medullaris*;—that part of the spinal cord which extends below the third sacral segment. In both cases the motor power of the legs is unaffected, and there is anæsthesia of the " saddle " area, with loss of sexual power and of control of bladder and rectum. Cauda lesions, however, are more often gradual in onset than conus affections, and are usually accompanied by intense sacral " root " pains, and the ultimate anæsthesia of a cauda affection is frequently preceded by cutaneous hyperæsthesia. A bed-sore is commoner in a medullary lesion than in a cauda lesion. Finally any " dissociation " of anæsthesia, such as analgesia or thermanæsthesia without tactile anæsthesia, points to a medullary lesion. If both cauda and conus are included in the same disease, the cauda symptoms mask the others.

Let us now turn to motor palsies resulting from affections *within the muscles* themselves. Some of these are really the effect of diseases of the intra-muscular fibrous tissue. This is the case in acute myositis where there is an interstitial inflammation within the muscle, often with a good deal of effusion, so that any sudden movement causes acute pain; tenderness on pressure is also present. Lumbago and the familiar " stiff neck " are common varieties, and their diagnosis usually presents no difficulty. Trichiniasis is a disease in which the muscle-fibres are invaded by the trichina spiralis. Here there is a history of the patient having eaten some meat, usually pork, which turns out to have been trichinosed. Within a day or two he developes gastro-intestinal symptoms and becomes feverish, with wide-spread muscular pains, rigidity, and stiffness, especially in the muscles of the limbs. In severe cases the pharynx, tongue, diaphragm, and even the laryngeal muscles may become affected. Together with this, there is a peculiar œdema of the face and eyelids, spreading thence to the limbs, and sometimes to the serous cavities. The blood contains a marked excess of eosinophile leucocytes, and the temperature is raised. The symptoms last usually four or five weeks.

Sometimes, when a patient has had his forearm fractured and put up in splints, the bandages may be applied too tightly and the blood-supply of the muscles is interfered with by the pressure. As a result there is swelling and pain in the hand, and unless the bandages be loosened, **ischæmic paralysis** may develope. At first the muscles of the forearm swell from effusion; later they become shrunken, hard, and contractured, the fingers being in a flexed position. Active movements are lost, and even passive movements are painful. The hardness and stiffness of the muscles and the absence of R.D., together with the normal condition of sensation, suffice, with the history of the case, to distinguish it from a neuritis.

But we also meet with cases of muscular weakness and atrophy localised in certain muscles, where the motor nerves and nuclei are healthy, but the muscle fibres themselves are primarily diseased. This group of diseases is called **muscular dystrophy, idiopathic muscular atrophy,** or **myopathy.**

Clinically we recognise two main classes of myopathy:—(1) those cases in which all the affected muscles waste from the outset; and (2) those in which certain muscles undergo a false enlargement before they ultimately become smaller—so-called pseudo-hypertrophic paralysis. But there is really no essential difference between these two varieties. Even in pseudo-hypertrophic cases, certain muscles undergo atrophy from the beginning, whilst in the others atrophy and enlargement may be combined in varying proportions.

Muscular dystrophy is a congenital disease. Not that the symptoms appear at birth, for the child is usually born apparently healthy. The age of onset of noticeable symptoms varies from two to sixty years, though most cases occur in childhood or youth.

The chief distinguishing features between myopathic atrophies and muscular atrophies of nuclear origin, spinal or bulbar, are as follows :—The age of onset is earlier, as a rule, in myopathy than in progressive muscular atrophy or amyotrophic sclerosis. Myopathy often runs in families; progressive muscular atrophy does not. The muscles affected are different in the two cases. In progressive muscular atrophy and amyotrophic lateral sclerosis, the wasting most commonly begins in the small muscles of the hands, attacking muscle-groups corresponding to segments of the cord. In myopathy the larger muscles are generally affected, whilst the small muscles of the hands escape. Amyotrophic lateral sclerosis tends to spread up to the bulbar nuclei, and to produce bulbar paralysis. Myopathy does not cause bulbar palsy. It is true that in one type of myopathy—the Landouzy-Dejerine—the face is affected, and that in pseudo-hypertrophic cases the masseters are sometimes enlarged; but myopathic palsy hardly ever affects the tongue, and never the larynx. In myopathy some of the diseased muscles may be enlarged, whilst others are wasted. Enlargement of muscles never occurs in nuclear disease. When a myopathic muscle voluntarily contracts, we may sometimes notice a ball-shaped swelling in the middle of the muscle, especially in the deltoid or quadriceps, which is due to the fact that the dystrophy is most marked at the extremities of the muscles. Fibrillary tremors, which are so common in progressive muscular atrophy, are rare in myopathy. Lastly, in myopathy, unlike amyotrophic lateral sclerosis, the deep reflexes are never increased. In fact, in myopathy the knee-jerk may disappear from wasting of the quadriceps.

The **pseudo-hypertrophic** variety of **myopathy** generally begins in childhood. It is the most rapidly progressive form of myopathy, and in most cases the patient dies before attaining adult age. Boys are affected four or five times as often as girls. The cases tend to run in families, and to select patients of the same sex in each family. Thus we often find several brothers affected whilst the sisters escape. But though the females generally escape themselves, they tend to transmit the disease to their male offspring, so that antecedent cases in a family are always on the mother's side. Children of the same woman by different husbands may suffer from the disease. It is therefore unwise to marry a widow, however young and charming, who has a pseudo-hypertrophic child. The symptoms of pseudo-hypertrophic paralysis are entirely motor. The first symptom which is noticed is that the child falls easily, gets up with difficulty, and cannot run or jump like other children. This weakness gradually increases until in a few years the patient becomes unable to stand or even to sit up in bed. The arms become weak later than the legs. Figs. 89 to 92 show two such patients. In them we notice the characteristic "pot-belly" and the lordosis when

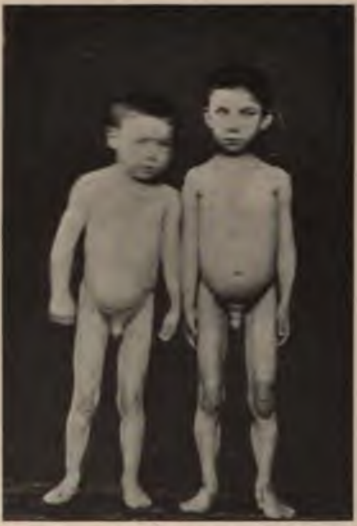

FIG. 89.—Pseudo-hypertrophic myopathy. Front view, showing enlargement of calves and thighs.

standing. The gait is wide-based and waddling, somewhat like that of the comic-opera sailor, and there is often a high-stepping action to clear the toes from the ground. But most characteristic of all is the way in which the patient rises from the floor when placed flat on his back. He first rolls round and gets on to his hands and knees. Then, keeping his feet wide apart, he drags his hands along the floor towards his feet until the knees are straightened. Then he shifts his hands up to his knees and proceeds to extend

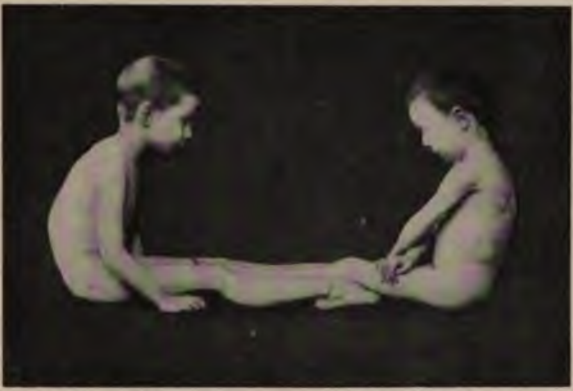

FIG. 90.—Pseudo-hypertrophic myopathy. Side view, showing absence of lordosis in sitting posture.

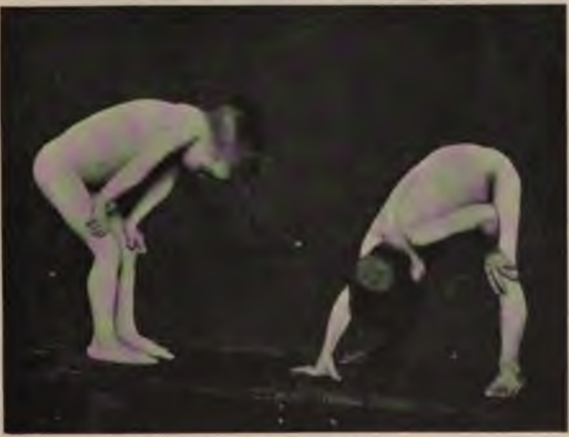

FIG. 91.—Pseudo-hypertrophic myopathy. Showing method of attaining the erect attitude.

the hip-joints by climbing up his thighs. When he has reached the upright position, he leans his trunk backwards to keep the hips extended.

In pseudo-hypertrophic myopathy certain muscles are specially enlarged and hard—*e.g.* the calf muscles and the infra-spinati. Other muscles may also become enlarged—*e.g.* the quadriceps, glutei, deltoid, biceps, and triceps. On the other hand, certain other muscles waste from the first, without any initial stage of enlargement. Thus the latissimus dorsi, teres major, and lower fibres of the pectoralis major are often absent, so that the folds of the axillæ are poorly marked, and if we lift up the child by the axillæ he tends to slip through our grasp.

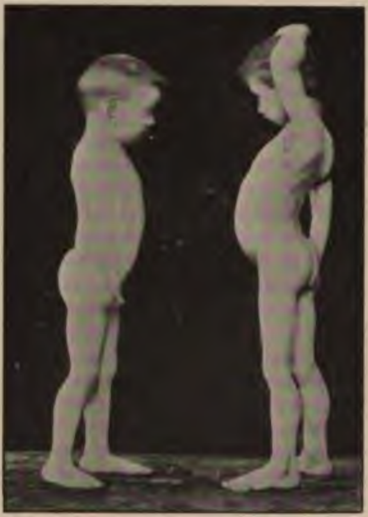

FIG. 92.—Pseudo-hypertrophic myopathy. Side view, showing lordosis when standing.

The enlarged muscles ultimately shrink and become smaller. As the calf muscles shorten, they produce a talipes equinus which still further hampers the walking powers. When the patient becomes bedridden, the contractures rapidly attain an extreme degree. As the quadriceps wastes, the knee-jerks disappear.

Primary atrophic myopathy is a less common type, and owing to absence of muscular enlargement, it is more likely to be confused with progressive muscular atrophy of spinal origin. At least three varieties have been recognised, according to the muscles which are first attacked. Thus we have (*a*) Erb's juvenile type, affecting the muscles of the shoulders and upper arms; (*b*) Landouzy and Dejerine's facio-scapulo-humeral type, where the atrophy begins in the face; (*c*) a type beginning in the lower limbs, chiefly in the anterior thigh muscles.

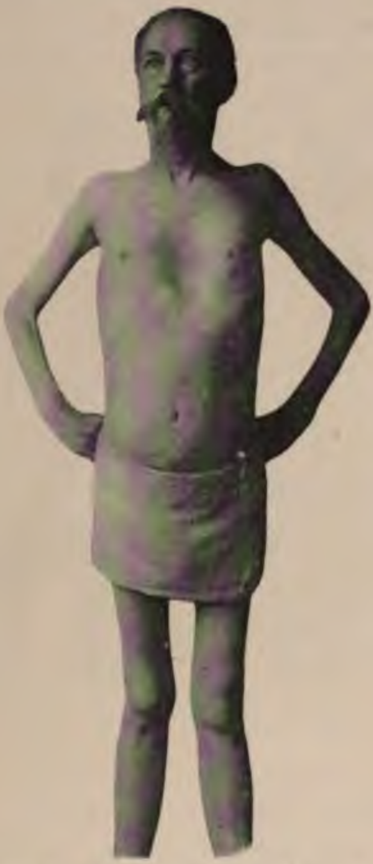

FIG. 93.—Muscular dystrophy. Erb's juvenile type.

In these atrophic varieties there is no striking preference for boys; both sexes are equally liable. The age of onset too is a little later, commonly between 15 and 35 years, except in facial cases, where the atrophy may come on in early childhood.

In *Erb's juvenile type* the atrophy begins in the large muscles of the upper arms and shoulders, especially the biceps, triceps, and supinator longus. Figs. 93, 94, and 95 are from such a patient, aged 47, who was a professional "living skeleton" in a travelling "freak" show. In his case the wasting was first noticed at the age of 19. In some instances, as in this particular case, the arms and legs are attacked about the same time. Or the disease may begin in the arms and spread to the legs. The deltoids and spinati often escape, even when the biceps and supinator longus are wasted, in this respect differing from cases of spinal origin, and the forearm muscles, except the supinator longus, generally escape too. From weakness of the glutei and quadriceps, the patient when rising to the standing posture may have to climb up his legs, as does a pseudo-hypertrophic case.

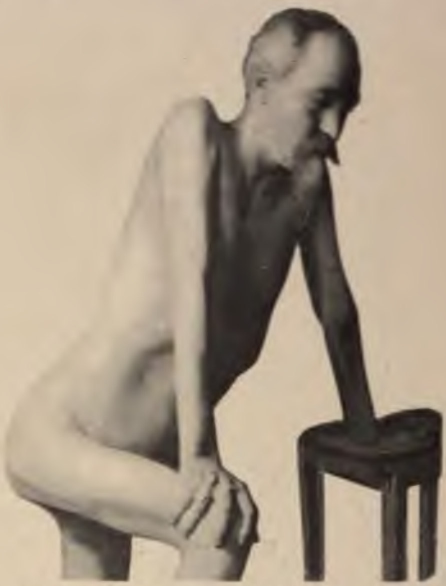

FIG. 94.—Muscular dystrophy. Erb's juvenile type. Patient endeavouring to rise to standing posture.

The *facio-scapulo-humeral* variety of *Landouzy-Dejerine* commences in early life, the facial muscles being earliest affected. The orbiculares oculorum and oris are weak, so that the patient cannot close his eyes tightly nor whistle. His lower lip droops and projects forwards, and his mouth habitually hangs open. The smile is peculiarly transverse and has a "forced" look, the angles of the mouth being drawn outwards but not upwards. The tongue, ocular, and jaw muscles are unaffected. Later the disease spreads to the scapular and upper-arm muscles, and finally to the spinal muscles and lower limbs.

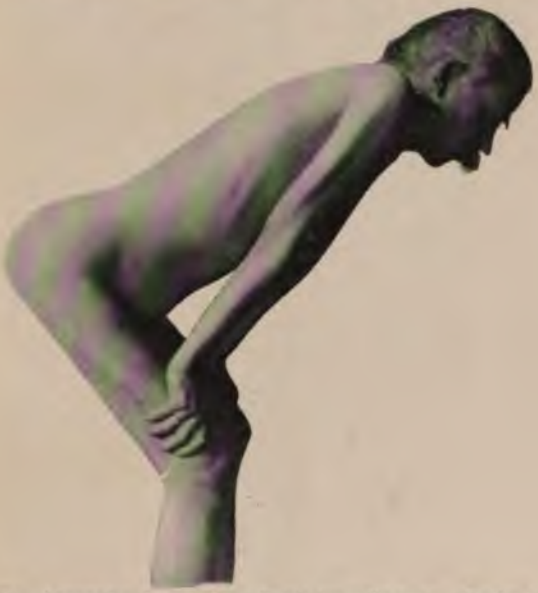

FIG. 95.—Muscular dystrophy. Erb's juvenile type. Patient rising to erect posture.

A third type of the disease, of which Figs. 96 and 97 are an example, begins in the legs, and attacks the arms later. In this patient the legs became weak at the age of 12. When she came under observation at the age of 28 she was still able to use her arms, which were contractured at the elbows, for knitting and to feed herself, though no longer capable of walking, owing to claw-foot.

Other Varieties of Muscular Wasting.—Wasting of muscles sometimes occurs as a secondary phenomenon in other diseases, where there is no implication of the spino-muscular motor neurone. Thus the muscles may become small from disuse, as may be seen in a limb which, owing to a fracture, has been confined for several weeks in splints. Disease of a joint is often accompanied by well-marked atrophy of the surrounding muscles. This arthritic muscular wasting specially affects the extensor muscles of the joint —for example, the quadriceps in disease of the knee-joint, the

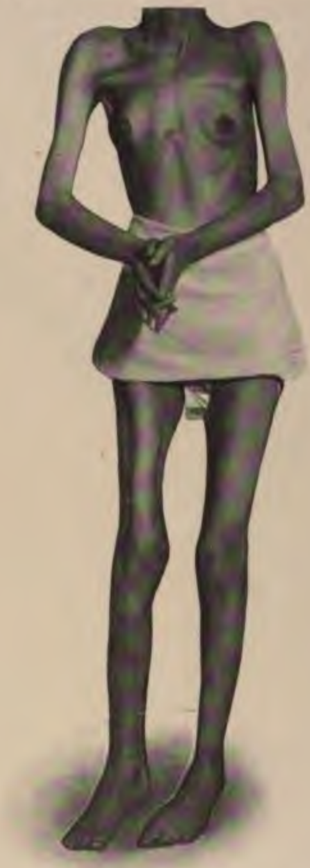

FIG. 96.—Muscular dystrophy; type beginning in lower limbs, but having advanced to upper limbs. Patient pressing hands together, to show atrophy of greater portion of pectoral muscles. Intrinsic muscles of hands unaffected.

interossei in osteo-arthritis of the hands, the deltoid in disease of the shoulder-joint. A degree of muscular wasting also occurs in the paralysed limbs in ordinary hemiplegia. Part of this may be the result of disuse, but there are other cases where the degree of wasting is excessive, and disproportionate to the paralysis. Lastly, we may meet with marked wasting of muscles in rare cases of hysterical paralysis (see Fig. 179, p. 326). But all these muscular wastings can be distinguished from that due to disease of the lower motor neurone, by the absence of electrical reactions of degeneration.

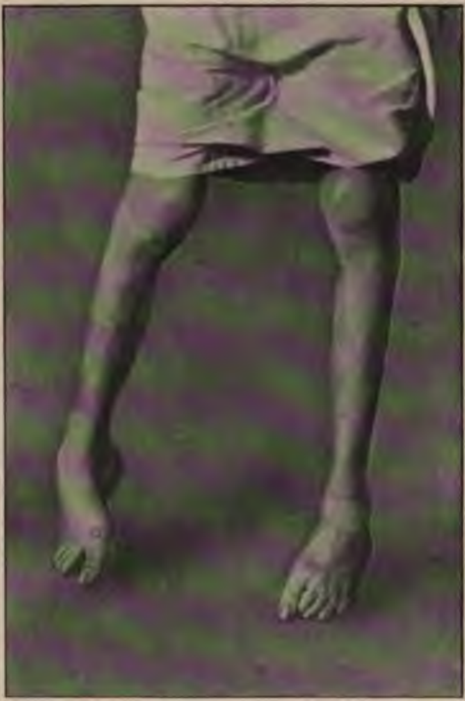

FIG. 97.—Muscular dystrophy. Same patient as in Fig. 96. Showing deformity of feet.

LECTURE XV

RECURRENT AND TRANSIENT PALSIES

THERE are certain forms of motor paralysis which, though transient, tend to recur again and again, the patient, in the intervals between his attacks, being able to execute all voluntary movements in a normal or almost normal fashion.

Of these diseases **myasthenia gravis** is the most serious. In myasthenia certain muscles become infiltrated with deposits of small round cells resembling lymphocytes, especially the striated muscles innervated by the upper cranial nerves, though sometimes the muscles of the limbs and trunk, and even the respiratory muscles, may become affected. The disease is characterised by the fact that the patient becomes, after very moderate exertion of the affected muscles, abnormally easily fatigued, and the affected muscles are, for the time, paralysed. The patient wakes up in the morning practically normal, but as the day goes on, certain muscles gradually become paralysed. Perhaps the muscles most often affected are the levators of the upper lids. Thus ptosis appears, often of unequal degree on the two sides (see Fig. 98). To this may be superadded an external ophthalmoplegia, generally incomplete. The facial muscles also become weak, and the patient has a peculiar " nasal " form of smile, in which the angles of the mouth are drawn upwards but very slightly outwards. The masseters and other masticatory muscles may also be affected, so that the patient cannot chew more than a few mouthfuls. The palate, tongue, and larynx may all be implicated, producing for the time the phenomena of bulbar palsy. In fact, the disease was formerly named " asthenic bulbar paralysis." To correct his ptosis, the patient may tilt his head backwards. But sometimes the neck muscles are affected, so that the head falls loosely backwards or forwards. In the limbs it is chiefly the large proximal muscles which are affected. But the most characteristic feature is the transitoriness of the paralysis, and its recurrence on slight exertion. Thus the patient can walk a short distance, but soon has to stop; he may speak a few sentences, and then his voice becomes weak and his articulation indistinct. In the affected muscles, the electrical reactions are altered and we have the "myasthenic reaction." Faradic shocks applied to the muscles at first produce brisk contractions, but on repeated stimulation the faradic excitability temporarily disappears. In contrast to the condition in true bulbar palsy, there is no muscular atrophy. The reflexes, deep and superficial, together with the sphincters, are unaffected. If the respiratory muscles become severely affected, the patient may die from respiratory failure.

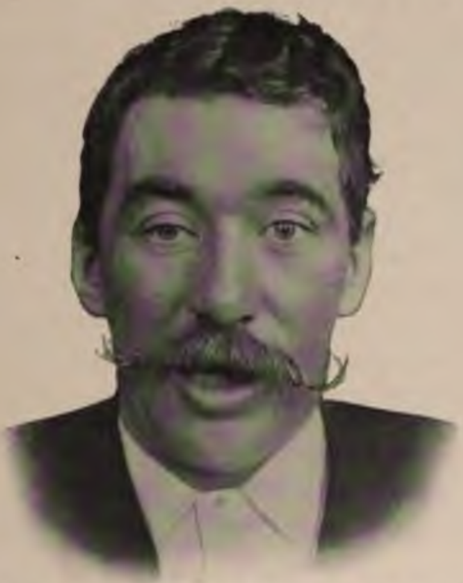

FIG. 98.—Myasthenia gravis in a man of 35. Marked weakness of masseters and of lower facial muscles; also right-sided ptosis. The patient is making a maximum effort to clench the jaws and to show the teeth.

Thomsen's disease, or "**myotonia congenita**," is a rare congenital affection of the voluntary muscles, which appears to be due to their possessing an excessive proportion of undifferentiated sarcoplasm and muscle-nuclei, and relatively too small a proportion of the anisotropic or fibrillar element. This latter contracts briskly, whereas sarcoplasm contracts very slowly. In Thomsen's disease, whenever the patient starts to perform a voluntary movement, his muscles are thrown into a state of tonic spasm, which gradually passes off, the muscles slowly becoming supple, until at length he can perform the movement, say that of walking, in a normal fashion. But if he stops and starts again, or if he tries to hurry his original speed, this stiffness reappears, and has again to be. as it were, worked off. The lower limbs are most commonly affected, but other muscles may also show the phenomena. Thus the patient's eyes may remain temporarily firmly closed after a cough or sneeze, his mouth may remain open after a yawn, and so on. The sensory functions and the reflexes are unaffected. But the affected muscles have a characteristic reaction to electrical stimulation. Their excitability is increased to both faradism and galvanism. To galvanism KCC is equal to ACC (instead of being greater, as in health), and the contraction set up, whether by galvanism or by faradism, is remarkably persistent, lasting for a time after the stimulus has ceased.

Eulenberg's disease, or "**paramyotonia congenita**," is a family affection somewhat like Thomsen's disease, in which a tonic spasm appears in certain voluntary muscles, more particularly in the face, so that the patient is unable, for a quarter of an hour or longer, to open his eyes or to speak. Less often the muscles of the limbs are affected. The condition, however, differs from Thomsen's disease in its immediate exciting cause. The tonic spasm of Eulenberg's disease is excited not by exertion but by cold, and is generally relieved by warmth. The two diseases, however, are closely allied, and have been observed coincidently in members of the same family.[1]

Another interesting variety of transient paralysis is that which is known as **intermittent limp**, or "**dysbasia angio-sclerotica**" ("claudication intermittent" of Charcot, or the "intermittirendes Hinken" of German authors). An analogous condition in horses, known as "spring-halt," has been familiar to veterinary surgeons for many years. The patient is most commonly a man of middle age, sometimes of gouty constitution, and very often has been an inveterate tobacco-smoker. The symptoms are very characteristic. At rest he feels no disability. But when he begins to walk, though he starts off normally, he soon begins to feel his legs tired, heavy, and painful. A cramp-like pain appears and gradually becomes intolerable, making him limp; and if he perseveres with his attempt, he finally becomes, for the time, totally unable to walk a single step. He rests, the pain and weakness pass off, only to return when he starts to walk afresh. If we examine such a patient during a paroxysm of temporary incapacity, we find his feet and legs cold, and perhaps purple or mottled red. But what is most characteristic of all is that the pulse in the feet, in the posterior tibial or the dorsalis pedis artery, is absent or nearly so. There are no sensory changes, the reflexes are normal, and, except during the paroxysms, the motor power of the limbs is unimpaired. All these phenomena appear to be the result of a temporary spasm of the arteries of the limb (the arteries themselves being frequently narrowed already by arterio-sclerosis), so that during walking an increased blood-supply to the muscles is not forthcoming, hence there result temporary anæmia, pain. and weakness in the affected muscles. In rare instances similar phenomena have been observed in the arms.

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[1] Delprat, *Deutsche med. Wochenschrift*, 1892, p. 158.

Amongst the transient paralyses we must also bear in mind the various craft-palsies, professional cramps or *occupation neuroses*, in which the limb is normal for all motor actions except one particular movement—often the one on which the patient's livelihood depends. The commonest variety is the so-called writer's cramp or scrivener's palsy, though we also meet with similar conditions in the cramp of pianists, violinists, telegraphists, typists, tailor's cutters, hair-cutters, hammer-men, cow-milkers, watch-makers, harpists, cigarette-makers. sempstresses, and so on. The cramp comes on, not during the period when the sufferer is learning his occupation, but after he has become an expert and requires to perform the particular skilled movement repeatedly for prolonged periods. For all other movements except that particular one, the limb is normal. Thus in writer's cramp the patient can use his hand normally for piano-playing or for grasping and using a heavy tool. This is because the weakness is not due to muscular but to cerebral fatigue. A professional cramp does not appear in its fully developed form at first, but passes through different stages of severity. In the slightest variety there is simply a degree of stiffness or spasm in performing the act, with a subjective sensation of pain and of intense mental discomfort and fatigue. In other cases a temporary paralysis appears when the patient attempts to write, so that the pen can no longer be held in the hand. In still more severe cases tremor is superadded to spasm in the affected muscles. The diagnosis is easy, inasmuch as the phenomena, whether spastic or paralytic or perhaps a combination of both, only supervene when one skilled motor action is performed, and the same muscles can be used for all other actions without pain, spasm, or weakness.

There is also a curious hereditary disease known as "**family periodic paralysis.**" This malady may run through several successive generations of the same family, attacking one or more members of the same generation. Males and females are both liable. The patient, who is otherwise apparently healthy, has attacks of flaccid paralysis of all four limbs, recurring irregularly without apparent exciting cause. The duration of a paroxysm varies from two or three days down to a few hours. The attacks generally come on during the night when the patient is in bed. He wakes up and finds himself more or less widely paralysed. First the legs are affected, later the arms, and last of all the muscles of the trunk and neck. The cranial nerves usually escape. In the limbs the paralysis starts proximally and the distal parts are affected last, so that the patient may still be able to move his toes and fingers when he has lost all power in the hips and shoulders. In severe cases the intercostal muscles may also be paralysed. Most striking of all is the fact that during these paroxysms of flaccid palsy the affected muscles, for the time, lose their excitability to faradic, galvanic, or mechanical stimulation, and all the reflexes in the affected limbs disappear. Sensation is unimpaired and the sphincters are unaffected. An additional point is that during the paroxysm, the left ventricle becomes temporarily dilated, as can be proved by percussion and occasionally by the appearance of a systolic mitral bruit. The muscular paralysis passes off in the reverse order to that in which it came on. The toes and fingers recover before the proximal muscles, and the muscles earliest attacked are the last to recover. The patient then remains apparently normal until his next attack, weeks or months later.

Amongst other transient affections we may mention those of **muscular cramp** and of **tetany**. Both of these are painful affections, the latter being most commonly met with in infants, whilst cramp is most often met with in healthy adolescents or adults who have been performing some unwonted prolonged muscular effort. The conditions are easily recognised, and both may temporarily interfere with the movements of the affected limbs. Tetany is generally bilateral, and has a characteristic posture of the hands and feet (see Fig. 24, p. 63).

Occasionally we see patients who complain that their legs suddenly give way in the street, causing them to fall. This may be due to various causes. For example, it is a not uncommon symptom in **tabes**. The tabetic patient often has hypotonia of the muscles about the knees and also deficient joint-sense, a combination of phenomena which may make him fall unexpectedly. In such a case the condition of the deep reflexes, the state of the pupils, and the other phenomena of tabes render the diagnosis easy. Other cases of sudden falling are due to **minor epilepsy**, where the patient has an attack of momentary unconsciousness during which he falls, but recovers consciousness at once and gets up again, not knowing why he has fallen. Here the diagnosis will depend on the observation of other epileptiform attacks, major or minor. We should particularly inquire for the occurrence of sudden pallor of the face or fixity of expression, indicating a passing attack of petit mal, too slight, perhaps, to produce a fall.

Sudden weakness of a limb may occur in **hysteria**, especially after some sudden emotional shock. Hysterical paralysis may persist for variable periods of time, varying from a few hours to many weeks or months. And the paralysis not uncommonly recovers as suddenly as it came on, sometimes under emotional or religious excitement, under hypnotic suggestion, or under energetic stimulation, electric or thermal, for example, that of a Pacquelin cautery. Hysterical paralysis never attacks a single muscle, but always a group of muscles. It is never accompanied by reactions of degeneration, no matter how much the hysterical limb may be wasted. We diagnose hysteria by a process of exclusion, noting not only the absence of certain signs of organic disease, but looking also for the presence of various hysterical "stigmata," to which we shall refer later.

But we must not forget that in many cases of apparently hysterical and transient paralysis in young women, the patient after one or more of such attacks may afterwards develope the signs of disseminated sclerosis. The onset of disseminated sclerosis may be indistinguishable from an attack of hysterical paralysis, the weakness of the limb in both diseases may be transient and may apparently clear up completely for a time. But a series of such attacks should always raise in our minds the suspicion of an underlying disseminated sclerosis and should make us give a guarded prognosis, especially if the patient has had more than one attack of weakness, not necessarily in the same limb. We should pay particular attention to the state of the optic discs. Early optic atrophy will exclude mere hysteria, so also will an extensor plantar reflex or anything approaching a true nystagmus. In fact, the disease which is most often wrongly diagnosed as hysteria is disseminated sclerosis.

LECTURE XVI

INCO-ORDINATION

A NEWLY-BORN child cannot co-ordinate the movements of its limbs. Certain co-ordinated vital actions, such as sucking, swallowing, respiration, &c., are performed well from birth, but in an infant's limbs the movements are mostly of an aimless restless character, with the exception, perhaps, of grasping movements of the hands. And even these latter differ from the co-ordinated grasp of later life, inasmuch as the infant's thumb is hardly used at all for opposition, and the flexion movement of the fingers occurs chiefly when some object comes in contact with the hand, the movement being reflex rather than voluntary.

The child only learns after long practice to use its muscles in such a fashion as to produce properly co-ordinated movements of the limbs. Walking, writing, swimming, the playing of any game, are all performed awkwardly at first. Skill is at last attained by frequent repetition, and once a co-ordinated action has been thoroughly learned, the effort for its performance becomes infinitesimal, so that in time it is performed more or less automatically.

Every co-ordinated action involves contraction not only of the so-called prime-movers but of their antagonists, and if these are not properly balanced, the attempted movement is awkward and jerky. This condition occurs in a number of diseases and is termed **ataxia** or **inco-ordination.** This means clumsiness, unsteadiness, or awkwardness in the performance of movements in a non-paralysed patient who was previously able to execute these movements in normal fashion.

In testing for ataxia of the upper limbs, we ask the patient to perform such an action as picking up a small object, say a pin, from a smooth surface. If he is ataxic, he fumbles during the attempt, or may perhaps pounce on the object in a sudden, jerky fashion. Another useful test is to ask the patient to lift a brimming glass of water to his lips and notice whether he spills it. Or we may ask him to touch rapidly the tip of his nose with each index finger in succession. If he is ataxic, his finger misses his nose by a greater or smaller interval, or on reaching it, the finger shows additional oscillatory movements. Other good tests for ataxia are to make the patient button or unbutton his coat or collar, or to write with a fine-pointed pen. In every case, we should observe whether the unsteadiness is increased or unchanged when the patient shuts his eyes. Slight degrees of ataxia due to sensory impairment may be noticed only when the patient is deprived of the help of his visual impressions.

In the case of the lower limbs we detect moderately severe ataxia by observing the patient's gait, asking him to walk "heel-and-toe" along a straight line, then to turn sharply and come back. In well-marked "locomotor" ataxia the gait is broad-based, the feet are lifted too high and the heels are brought down with a stamp. In cerebellar disease, on the other hand, the patient reels or lurches along, being especially unsteady when turning round (see later, "Postures and Gaits"). To detect ataxia of a single lower limb, we ask the patient to place one heel on the opposite knee, or to trace with one foot a circle or some other pattern on the floor, or we ask him when lying down to touch with his toe our own finger held in the air. And here also, in ataxia of the lower limbs, we should always note whether the patient's unsteadiness is increased or unchanged by shutting the eyes. In tabetic ataxia of the lower limbs, the unsteadiness is increased when the eyes are shut. Romberg's sign consists in the tendency of a patient, who can stand steadily with the eyes open, to fall when he closes them. Thus an early sign of tabes is the so-called "wash-basin" sign, where the patient falls into his basin when washing his face. A minor degree of Romberg's sign can often be detected in the earlier stages of tabes, where the patient is still able to stand with the eyes shut, but the tendons on the dorsum of the foot are seen to exhibit irregular restless contractions, the so-called " danse des tendons."

Ataxia of the bulbar muscles has already been referred to (see Dysarthria, p. 92). As to ataxia of the muscles of the trunk, its diagnostic significance is less than that of the limbs, inasmuch as by the time that the trunk muscles are recognisably ataxic, the limbs already show very marked unsteadiness. Ataxia of the head and trunk muscles is evidenced by swaying movements when the patient sits up.

Having detected ataxia in the movements of any limb, we must always determine the condition of the sensory impulses from that limb, testing not only the various forms of cutaneous sensibility but also the deep sensations, especially the kinæsthetic sense and, still more important, the joint-sense. Finally, we must determine the condition of the deep reflexes, noting their exaggeration as in disseminated sclerosis, or their abolition as in tabes or in Friedreich's ataxia. We also note the type of plantar reflexes.

The commonest variety of ataxia is that due to deficiency of peripheral impressions—not so much from the skin as from the deeper structures, the muscles and joints. Thus in lesions of *peripheral sensory nerves* an anæsthetic limb is often ataxic. Division of the *posterior roots* of the brachial plexus, the anterior roots remaining intact, causes marked ataxia of the upper limb. In fact such a patient may be totally unable to move the limb if his eyes are closed, so that he no longer has the guiding influence of vision. Degeneration of the posterior roots and posterior columns, as in *tabes*, produces marked ataxia. A tabetic patient is ataxic mainly because of deficiency of afferent impressions, more especially from his muscles and from his joints. If the muscle-tonus is lost and the muscles are hypotonic, as in many cases of tabes, the patient has, as it were, to " pull in the slack " before the muscles come into proper play, thereby making the movement jerky and inharmonious. When the joint-sense is impaired, he has to perform a maximal movement in order to get the sensation of having moved the joint at all. Thus in walking when he lifts his leg, owing to want of proper joint-sense, he throws the limb up with abnormal suddenness and to an unnecessary height in order to gain the sensation of flexion of the joint. He then stamps it down with excessive emphasis to make sure that it really is extended. Such a patient with ataxia of sensory origin compensates for the deficiency of sensory impressions from his limbs by means of his vision. When he is watching his partially anæsthetic limbs, he can control them better. Hence if he closes his eyes, the regulating influence of vision is lost and he becomes much

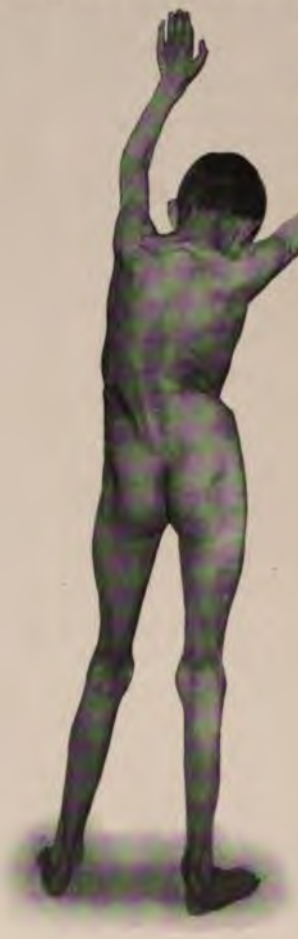

FIG. 99.—Friedreich's ataxia. Showing scoliosis.

more ataxic. This is the explanation of Romberg's sign in tabes.

Ataxia similar to that of tabes also occurs in other organic cord lesions implicating the posterior columns, as for instance in *tumours* or *chronic sclerosis of the posterior columns*, whether combined or not with lateral sclerosis—*e.g.* some cases of disseminated sclerosis or spinal syphilis. In such diseases the other signs and symptoms guide us to a correct diagnosis.

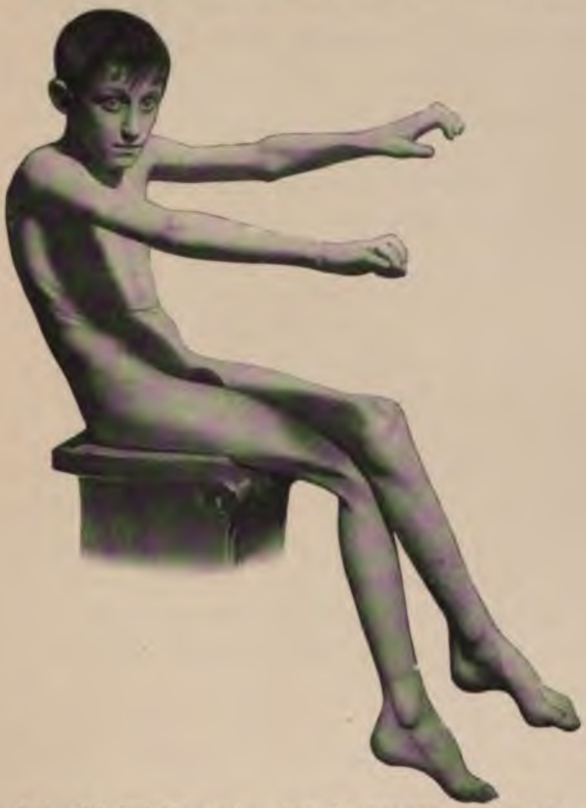

FIG. 100.—Friedreich's ataxia. Showing pes cavus and manus cavus.

But a patient may be ataxic when all his peripheral sensations are normal. This occurs in the different varieties of cerebellar ataxia. Let us take, for example, *Friedreich's ataxia*, a developmental disease affecting mainly the afferent tracts in the cord leading upwards to the cerebellum, which is the centre for automatic co-ordination. In Friedreich's ataxia the patient becomes ataxic, as in tabes, but with the cutaneous and joint-sense intact. The ataxia is unaffected by closing the eyes. The age of the patient, who is commonly an adolescent, the presence of scoliosis, of manus cavus, and of pes cavus (Figs. 99 and 100), the normal pupillary reactions, the presence of nystagmus, the absence of lightning pains or of bladder trouble, all serve to distinguish this disease from tabes, though in both diseases the deep reflexes are absent. The plantar reflexes in tabes are flexor in type, whilst in Friedreich's ataxia they are of the extensor variety.

Ataxia also occurs in certain localised lesions of the *medulla oblongata*. Thus, for example, a unilateral lesion may interrupt the fibres of the direct cerebellar tract and thereby interfere with co-ordination of the homolateral limbs. Such a lesion, which usually interrupts at the same time the fibres of Gowers' tract, may extend inwards to implicate the inter-olivary arcuate fibres and the fillet, together with the nuclei of the lower cranial nerves. It produces a characteristic syndrome, known as the *unilateral bulbar syndrome of Babinski and Nageotte.*[1] The symptoms are as follows :—From interruption of the cerebellar afferent fibres there is ataxia of the homolateral limbs. From interference with the tract for pain and temperature we have analgesia and thermanæsthesia of the opposite side of the body (see Fig. 9, p. 13). From interference with the oculo-pupillary centre in the bulb there is myosis and pseudo-ptosis (see later, "Cervical Sympathetic Paralysis," p. 287), and there may be difficulty of deglutition due to affection of the cranial nerve nuclei. If the lesion extends forwards to implicate the pyramid of the same side, there is hemiplegia of the contra-lateral arm and leg.

Marie's hereditary cerebellar ataxia is very similar to that of Friedreich's ataxia, but there is no loss of knee-jerks, and the age of onset is somewhat later. Cerebellar ataxia also occurs in cerebellar tumours, in vascular lesions of the cerebellum, in disseminated sclerosis affecting the cerebellum or its peduncles, in cerebellar abscess, and in encephalitis of the cerebellar cortex, a disease of febrile onset, which is sometimes met with in children.

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[1] *Revue Neurologique*, 1902, p. 358.

Cerebellar ataxia differs in several important respects from the ataxia due to deficient afferent impressions. It is chiefly evidenced in the cerebellar gait, which has a staggering, reeling character, like that of a drunken man, but without the stamping of the true tabetic. We recognise several factors in the production of cerebellar ataxia. Part of the ataxia, especially that of the trunk muscles, is due to vertigo, the patient being unsteady in his gait because he feels giddy. A cerebellar gait therefore occurs in many cases of labyrinthine disease. If a cerebellar patient lies down, his vertigo is diminished and there being no longer any effort required to maintain equilibrium, his movements are much less unsteady. Another factor in cerebellar ataxia is the existence of the muscular hypotonia which we often meet with in cerebellar disease; this hypotonia, in unilateral cerebellar lesions, is more marked in the limbs of the affected side. The third and most important factor in cerebellar ataxia is the want of the co-ordinating influence of the cerebellum on the cerebral motor cortex. This connection is a crossed one, the right cerebellum being connected with the left cerebral cortex, *viâ* the right superior cerebellar peduncle and the left red nucleus. Some tumours of the frontal lobe have produced cerebellar ataxia, probably owing to indirect interference with the functions of the contra-lateral side of the cerebellum. True cerebellar ataxia, unlike the ataxia of tabes dorsalis, is uninfluenced by closure of the eyes.

Ataxia also occurs in some affections of the higher cerebral centres. For example there are certain *toxic* affections in which the patient becomes ataxic. The most familiar variety is the ataxia of acute alcoholic intoxication. Part of this ataxia may possibly be due to cerebellar intoxication, but a large part of it is cerebral, as evidenced by the "mental ataxia," the disordered articulation, &c. The temporary ataxia of *fatigue*, of writer's cramp and of other occupation-neuroses is also probably of cerebral origin, so also is the transient ataxia which sometimes follows enteric fever or other exanthemata.

There are certain diseases of the cerebral cortex, in which ataxia is distinct. In *chorea* the patient not only exhibits spontaneous involuntary movements, but he also has distinct ataxia on voluntary movement. And in organic *monoplegia* or *hemiplegia*, especially when slight in degree and amounting merely to paresis, distinct ataxia often exists in the paretic limbs. Sometimes ataxia of the limbs precedes an attack of hemiplegia—*pre-hemiplegia ataxia*, especially in threatened softening from arterial thrombosis. More often the ataxia appears during convalescence from a slight hemiplegic attack—*post-hemiplegic ataxia*, where the patient has to learn the process of co-ordination again in his paretic limbs. This form of ataxia must be distinguished from athetosis, the involuntary slow writhing movements of the limbs which occurs in old and severe cases of hemiplegia, especially of infantile hemiplegia.

Ataxia is one of the most striking signs of *disseminated sclerosis*, where the unsteadiness of the limbs is often associated with a superadded coarse oscillatory tremor—the so-called intention-tremor. How much of the unsteadiness in disseminated sclerosis is due to cerebral and how much to cerebellar disease it is difficult to say in any individual case. The tremor of hemiplegic distribution which is observed in the limbs in cases of *lesions of the red nucleus or rubro-spinal tract* (see p. 72) is present at rest, but becomes exaggerated on voluntary movements, rendering them ataxic. In this respect it contrasts with the tremor of paralysis agitans, which can usually be controlled to permit of the performance of a voluntary movement.

Finally we may meet with ataxia of the most varied types in *hysteria*. Here the affection is probably one which implicates the highest psychical centres. The diagnosis of hysterical ataxia rests on the presence of other stigmata of hysteria, together with the absence of evidence of organic disease. Sometimes hysterical ataxia is associated with "cortical" anæsthesia of the affected limb. In such a case the patient may be able to move the limb normally with her eyes open, but when they are closed ataxia appears. This does not necessarily occur in every hysterically anæsthetic limb, for in many cases profound anæsthesia may be present without ataxia. The diagnosis of hysterical ataxia, however, seldom presents much difficulty to a careful observer. The disease which is most often mistaken for hysteria is disseminated sclerosis in its earlier stages. In both diseases we may have a history of transient weakness of a limb, apparently clearing up completely for a time. But in disseminated sclerosis there are evidences of organic disease in the form of pallor of the optic discs, nystagmus, alterations of the abdominal and plantar reflexes, sphincter trouble, and so on.

LECTURE XVII

POSTURES AND GAITS

Postures.—In health the posture of the body and of its various members is determined partly by gravity, partly by the relative strength of the muscles at the various joints. And therefore, inasmuch as the flexor muscles of our limbs are usually more powerful than the extensors, the ordinary posture of the limbs at rest, generally speaking, is one of slight flexion. This is easily verified by observing a sleeping child. In the erect attitude the co-ordinating action of the cerebrum and cerebellum come in. The minor varieties of posture in different healthy individuals, which we learn to recognise as part of each man's personal characteristics, are largely the result of differences not only in muscularity but of habit. The pose of a powerful, muscular man is widely different from that of a thin, debilitated invalid. Moreover, if from exercise or want of exercise, certain groups of muscles are more or less developed than the normal, the posture is further modified, even in health. For example, we all know the characteristic straddling gait of the professional jockey.

Similar principles apply to those cases of organic disease where certain muscles or groups of muscles are affected by paralysis. Paralysed limbs gradually assume characteristic postures. And these **postures of organic disease** are not matters of haphazard, but are determined by anatomical rules.

Firstly, if the muscular paralysis be the result of a *lower motor neurone lesion*, in the anterior cornua, anterior nerve-roots, nerve trunks, or muscle-fibres, the paralysed muscles become wasted. Their unopposed non-paralysed antagonists slowly become contracted, and fix the limb in a certain definite posture which is best demonstrated if the patient tries to throw the paralysed muscles into action, in which case the antagonists contract alone.

For example, Fig. 101 is a photograph of a woman who had a localised lesion in the anterior cornua of the cord at the level of the fifth cervical segment. Amongst the chief muscles supplied by the anterior cornua at that level are the deltoid, biceps and supinators. These muscles underwent atrophy and their unopposed antagonists became contracted. As a result we see that from paralysis of the deltoid and contraction of its opponents the shoulder is adducted, similarly from paralysis of the biceps the elbow is extended by its opponents, and from paralysis of the supinators the forearm is hyperpronated by their antagonists. This posture is characteristic of a cord lesion at the fifth cervical segment.

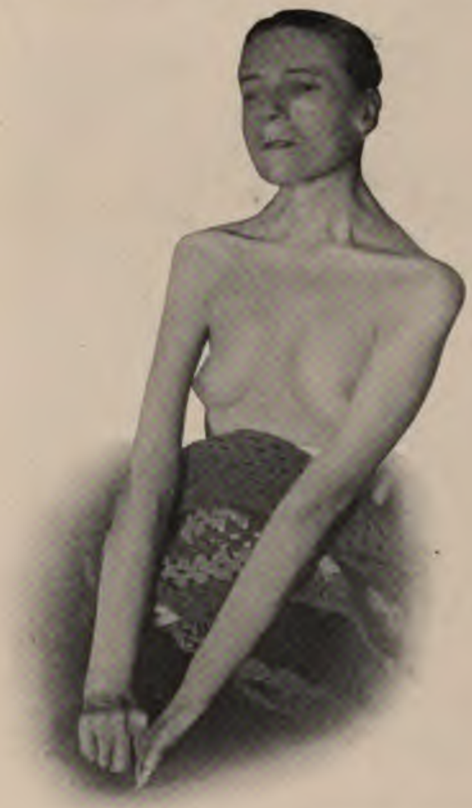

FIG. 101.—Chronic myelitis of fifth cervical segment, with atrophic paralysis of deltoid, biceps, and supinators.

Fig. 102 shows the posture assumed in a case of paralysis of the external popliteal nerve. This nerve was divided by a bullet-wound in an officer's right leg. In addition to anæsthesia corresponding to the cutaneous distribution of the nerve, the figure shows the presence of foot-drop due to paralysis of the anterior tibial group of muscles, with unopposed contraction of the calf muscles.

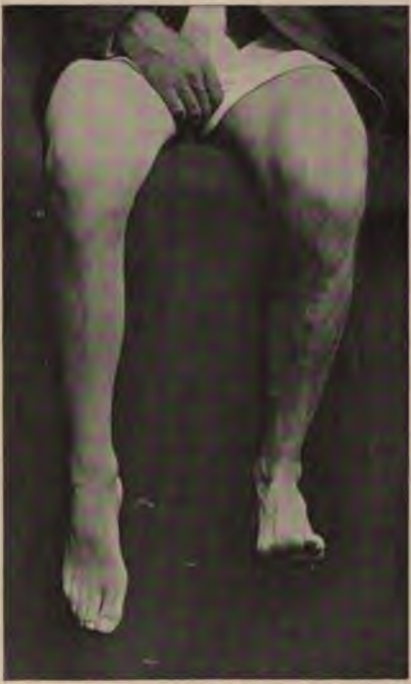

FIG. 102.—Paralysis of external popliteal nerve of right leg, the result of a bullet-wound. Showing muscular atrophy and drop-foot.

Here, of course, the action of gravity is a factor as well, the weight of the foot tending to increase the foot-drop.

Fig. 103 shows the posture in a case of paralysis of the musculo-spiral nerve. The patient is trying to extend both his wrists. On the paralysed side we notice the atrophy of the supinator longus and the paralysis of the extensors of the wrist and fingers, also the characteristic swelling on the dorsum of the hand, probably bursal in origin, which appears in long-standing cases of drop-wrist.

Secondly, if the paralysis be due to an *upper motor neurone lesion*, the law which determines the posture is different. We no longer have atrophic paralysis, limited to a particular muscle or muscles as in a lower motor neurone lesion. Instead, there gradually developes a spastic paralysis, in which all the muscles of the affected limb or segment of a limb are more or less paralysed. The muscles which are normally more powerful are therefore placed at an advantage during the process of spastic contracture, and a posture results which is an exaggeration of the normal attitude at rest, so that, for example, in a chronic hemiplegia the familiar posture of the upper limb is that of flexion with pronation, that of the lower limb being one of slight flexion at the hip and knee, with extension and inversion of the ankle, and a tendency to dorsiflexion of the toes (see Figs. 104 and 105).

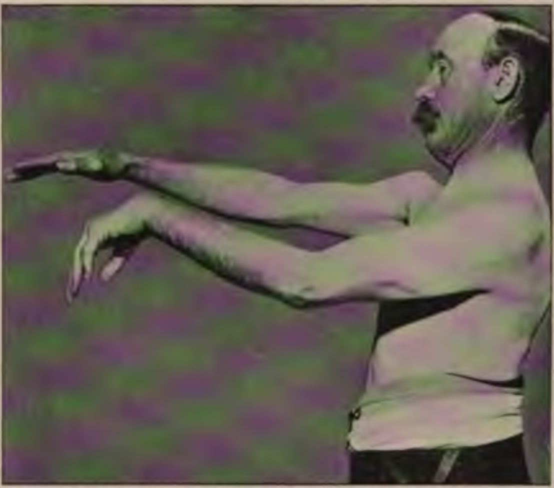

FIG. 103.—Left-sided musculo-spiral paralysis. Showing drop-wrist and atrophy of supinator longus.

This spastic posture in hemiplegia does not come on at once. There is an initial flaccid stage, lasting several weeks or even months, before the spastic rigidity sets in. (In a small number of cases the hemiplegia may remain permanently flaccid.) But even during this flaccid stage the postures of the hemiplegic limbs are often different from those of the unaffected side at rest. One of the most characteristic signs is an apparent broadening of the lower limb (Heilbronner's[1] "breites Bein"). This consists in an outward rotation of the lower limb at the hip-joint, due to its own weight when in the recumbent posture (analogous to the displacement which occurs in fracture of the neck of the femur). The thigh, therefore, when looked at from the front, appears broader than on the healthy side. Moreover, the flaccid paralysed muscles fall back by their own weight, and on transverse section the thigh forms a flattened oval instead of an approximate circle as on the healthy side. This apparent broadening of the paralysed thigh can

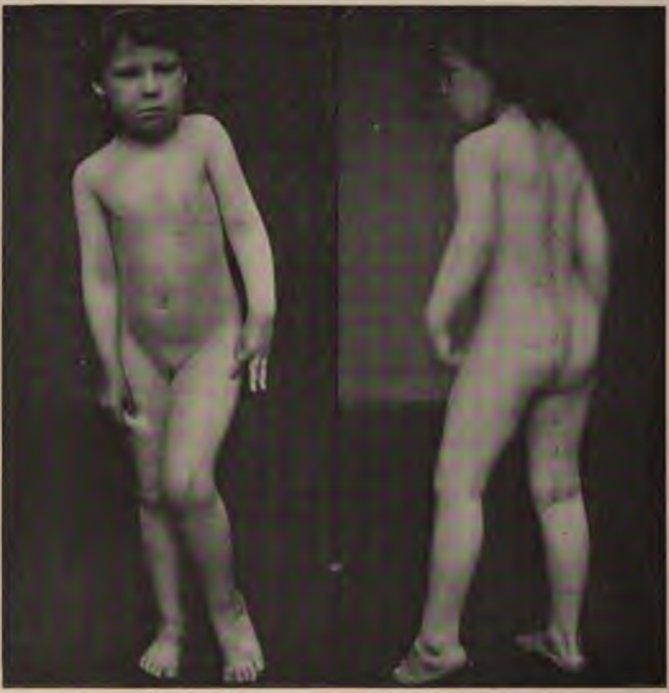

FIG. 104. FIG. 105.

Case of left-sided infantile hemiplegia in a patient aged 7 years. Onset of hemiplegia five years previously. Showing posture of limbs. In Fig. 104 note athetosis of left fingers. In Fig. 105 note inversion of ankle and drawing up of heel.

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[1] *Deutsche Zeitschrift für Nervenheilkunde*, Bd. 28, 1904, p. 1.

be well seen if the patient be seated on a hard, flat seat. It does not occur in hysterical hemiplegia.

In **functional paralysis** the conditions are different. Hysteria, it is often said, may simulate organic disease—thus we may have a functional hemiplegia, paraplegia, or monoplegia. But if we examine carefully we usually find that this similarity is more or less rough and inaccurate. And why? Because hysterical contractures are not governed by anatomical rules ensuring the preponderance of the stronger muscles. Hysterical contractures usually present some points in which they differ from the postures

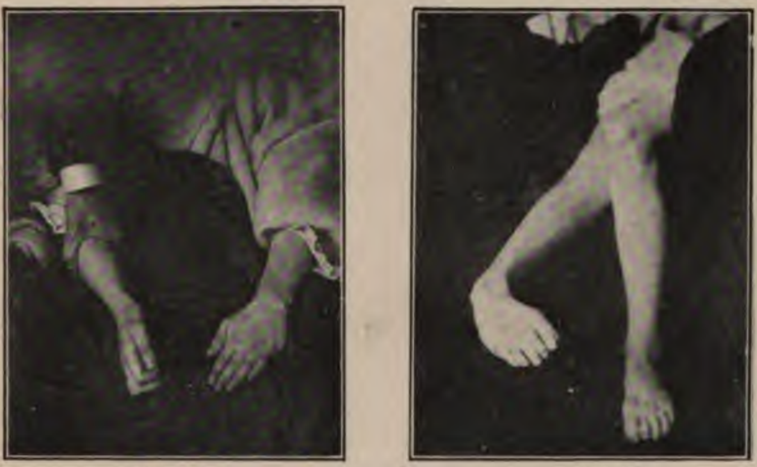
FIG. 106. FIG. 107.

From a case of right-sided hysterical hemiplegia in a left-handed patient. Showing contractures of hand and foot. The right forearm was rigidly supinated, but is being passively rotated inwards by the physician, to show the posture of the hand.

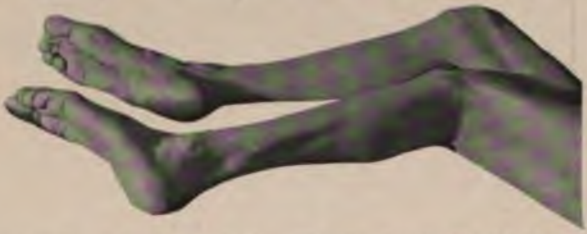
FIG. 108.—Functional paraplegia. Showing longitudinal fold of soles.

of genuine organic lesions. Figs. 106 and 107 are from a case of functional hemiplegia, in which the contracture alone was enough to distinguish it from an organic case. Instead of the usual flexed and pronated posture of the upper limb, we observe that the elbow and wrist are extended, the forearm is supinated, and the fingers are half-bent in a hook-like fashion, and in the lower limb the inversion of the ankle is overdone, out of all proportion to the ordinary equinus position. The contracture in this patient appeared suddenly, as is so often the case in hysteria, unlike the gradual development of an organic contracture. Fig. 108 shows a peculiar contracture of the soles in another case of hysterical para-

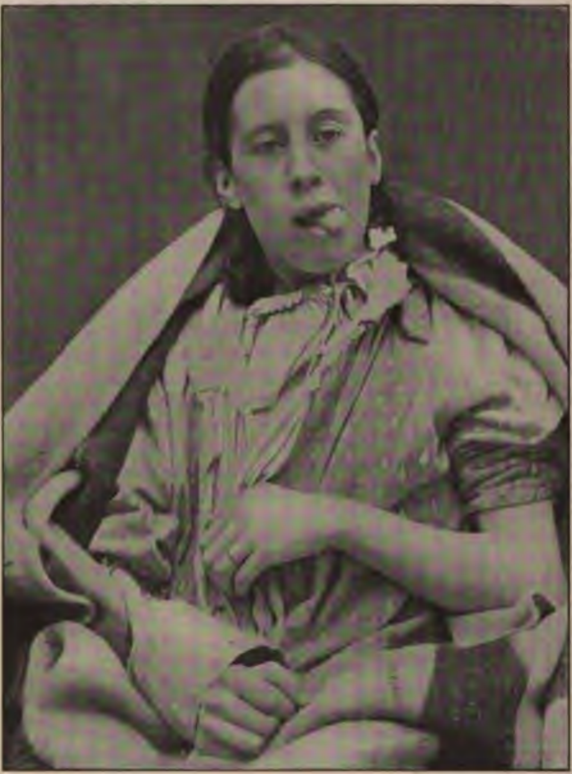

FIG. 109.—Left-sided hysterical hemiplegia with glosso-labial spasm on protrusion of tongue. The left upper limb was also rigidly contracted at the shoulder and elbow, the hand being flaccid.

plegia, in which the feet had a fold running longitudinally along the soles, totally unlike any organic contracture.

Fig. 109 shows a case of left-sided hysterical hemiplegia, in which the contracture of the upper limb differs from that of organic disease; for whilst the shoulder, elbow, and wrist are rigid, the fingers are flaccid—a combination which does not occur in organic hemiplegia. We also observe that this patient has glosso-labial hemispasm. When she protrudes her tongue it deviates considerably to the paralysed side, whereas in most cases of organic hemiplegia, if there is tongue deviation, it is usually towards the unaffected side. But what is still more important to note is that, when the tongue is protruded, the face on the paralysed side goes into a state of spasm, so that the left naso-labial fold is deeper than on the healthy side, a condition which is the reverse

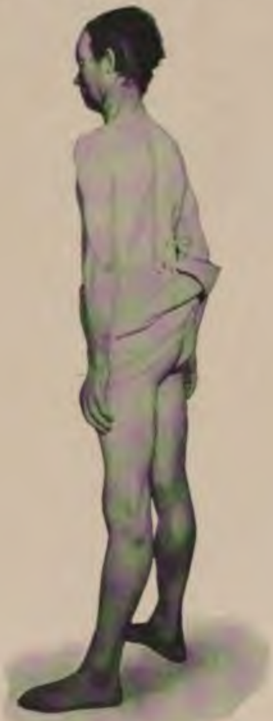

FIG. 110.—Hyper-extension of knees in a case of tabes—"*genu recurvatum*."

of what we find in organic hemiplegia. Glosso-labial hemispasm such as this is not a common sign, but when it occurs it is pathognomonic of hysteria.

Hysterical contractures often, but not always, disappear during sleep or during deep anæsthesia. If the contracture has persisted for months, adhesions may form in the joints, so that even under an anæsthetic the contracture may not completely relax, and we may have to break down the adhesions forcibly.

The postures of organic paralyses, then, whether supra- or infra-nuclear, are definite and comparatively simple, being governed by the anatomical rules we have mentioned. Hysterical contractures, on the other hand, being under no such restrictions, may assume the most varied and weird appearances, examples of which might be multiplied indefinitely.

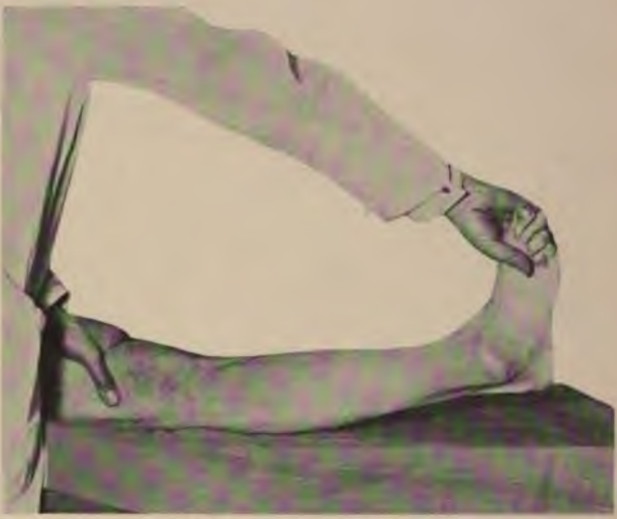

FIG. 111.—Tabetic hypotonia of popliteal muscles.

In many cases of tabes dorsalis there is marked deficiency of muscular tonus, a condition known as *hypotonia*. This slackness of the muscles has a marked influence on the patient's postures. Thus, for example, when the hamstrings and sural muscles behind the knee are hypotonic (see Fig. 110), the joint becomes hyperextended when the patient stands—the so-called "genu recurvatum"—unlike the knee-joint of a healthy individual, in which, however strongly the knee be extended, there always remains a concavity behind. This hypotonia of the popliteal muscles produces another very characteristic sign of tabes, which is that when the patient lies in bed with the knee extended, the heel can be passively raised whilst the back of the knee remains in contact with the bed (see Fig. 111). Hypotonia of the muscles of the trunk and lower limbs in tabes may permit of the patient assuming the most extraordinary postures without

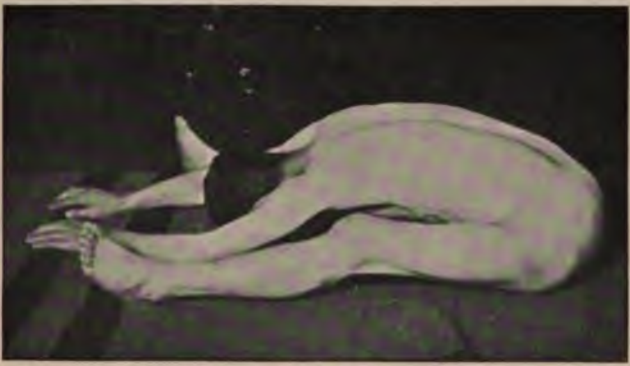

FIG. 112.—Tabetic hypotonia of gluteal and hamstring muscles.

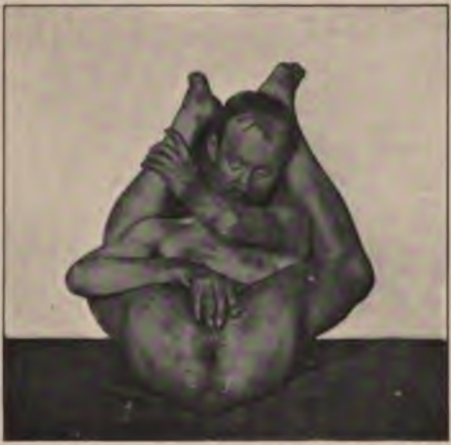

FIG. 113.—Tabetic hypotonia of muscles of lower limbs and trunk.

pain—postures which are impossible to any ordinary individual who is not a professionally trained contortionist (see Figs. 112, 113 and 114).

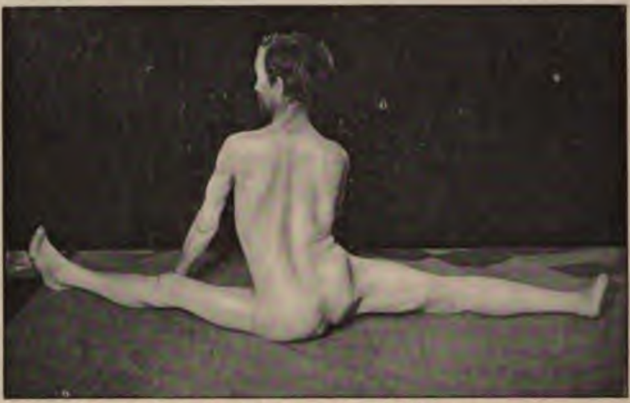

FIG. 114.—Tabetic hypotonia of adductor muscles.

Gaits.—Unlike some of the lower animals—for example, the chicken or the lamb—the human infant at birth cannot stand or walk. It is not until the child reaches the age of about eighteen months that he begins to walk. First he learns to stand, and then after repeated efforts he succeeds in walking. During the process of learning, he reels and falls about just like an adult with cerebellar disease.

We do not all of us walk exactly in the same fashion. Even healthy individuals show minor differences in gait. An old man walks differently from a youth, a soldier differently from a sailor, and a woman advanced in pregnancy differently from a young maiden. The wearing of ordinary boots with artificial heels also modifies the gait, so that in health the first part of the boot to show signs of wear is usually the back of the heel on the outer side. Ladies' high-heeled boots modify the gait still more, throwing the weight of the body unduly forwards towards the heads of the metatarsal bones.

When testing the gait in cases of nervous disease it is advisable to have the patient's lower limbs well exposed, and without boots. To get a good view of the limbs it is well to have the minimum of clothing on the patient. A good plan is to make the patient don some light garment like a shirt, whose tail is pulled forwards between the legs from behind and pinned in front, thereby leaving the upper limbs free. We ask the patient to walk straight away from us towards some given point, then to turn round and come back.

In a *normal gait* the limbs are moved forward easily, the feet neither scraping the ground whilst being lifted, nor being unduly stamped down as they descend. The forward-moving or "active" leg is the one which carries the weight of the body. The trunk therefore leans a little towards the corresponding side during the "active" phase of each limb. This trunk movement is attained partly by the action of the gluteal muscles, partly by the sacro-lumbar muscles of the same side. A short, thick-set person with a broad pelvis tends to have a more "waddling" gait, as the weight of his trunk is shifted across from one side to the other. And when the gluteal muscles are weak, the patient waddles excessively, in his efforts to throw his weight to each side alternately.

The gait is altered in various diseases. Thus it may become spastic, ataxic, reeling, high-stepping, and so on.

A *spastic gait* occurs in lesions of the pyramidal tract—for example, in hemiplegia, in diplegia, or in spastic paraplegia. In *organic hemiplegia* the active forward-projection of the limb is especially difficult on the paralysed side, and the weight of the body has to be carried forwards by the aid of the other side *plus* gravity, unlike the normal gait where the weight is carried entirely by the "active" advancing leg. Thus when the hemiplegic leg ought to be in the "active" phase, it is not properly flexed at the knee or ankle, nor is it actively pushed forwards but it merely dangles forward like a pendulum, not directly forwards, but swinging in a circular fashion round the opposite hip as on a pivot. And when the "active" phase of the non-paralysed limb occurs, the hemiplegic foot (owing to weakness of the peronei and dorsiflexors of the ankle, with over-action of the invertors and calf muscles) stays on the ground too long, thereby scraping the front part of the foot, especially the ball of the great toe; the stride of the paralysed limb is therefore shorter than on the healthy side. In a case of *spastic diplegia*, or double hemiplegia, the patient is unable to project either leg forwards in the ordinary way, but has to jerk each forwards in turn with a circular swing, so that, in a well-marked case, not only does the patient take abnormally short steps, catching the ball of each foot on the ground, but from the circular swing of the limbs, together with their adductor spasm, they cross alternately in front of each other, producing the cross-legged or "scissor" gait (see Fig. 115). Meanwhile the trunk and upper limbs make violent jerky movements, swinging the body from one side to the other. In ordinary *spastic paraplegia* from cord disease, there is not the cross-legged gait of diplegia, but the patient moves stiffly along, taking abnormally short steps, the front part of the

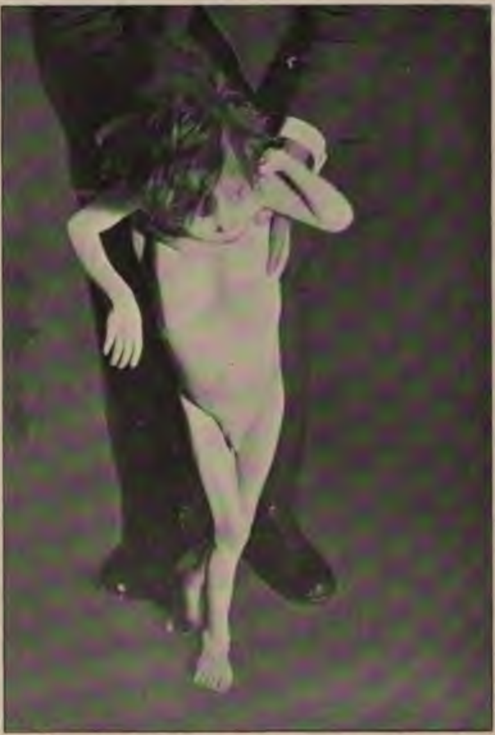

FIG. 115.—Spastic diplegia. Showing "scissor-gait.

feet clinging to the ground, thus wearing out the toes of the boots. Meanwhile the tendency to ankle-clonus causes a "trepidation" of the whole body in severe cases, from tremor of the feet. Such a patient stumbles over the slightest obstacles.

The *gaits of hysteria* are of the most varied types. For example, in hysterical hemiplegia, the patient often pushes the paralysed foot along the ground as if on a skate, or drags it helplessly along with its dorsum resting on the ground, as seen in Fig. 116. If this is bilateral, the patient is totally unable to walk or stand. Or the foot may be held in a position of talipes calcaneus when walking, even when no such posture is present at rest; or one lower limb may be acutely flexed at the hip and knee, so that the patient has to use crutches. Or again, the patient may have a zigzag gait, or he may throw one leg about with a wild flourish before bringing it to the ground. *Astasia-*

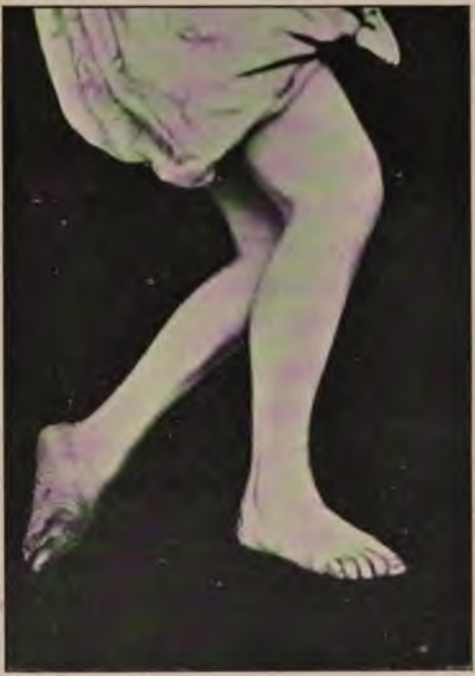

FIG. 116.—Hysterical paraplegia, with dragging of the left foot.

abasia is the term applied to a condition in which a hysterical patient is unable to stand or walk, although capable in the recumbent posture of performing all movements of the lower limbs normally. The varieties of hysterical gaits are practically unlimited, but every one of them differs in some respect from the gait of organic disease.

The *side-gait* (Schüller's "Flankengang") is a useful means of diagnosing between organic and hysterical hemiplegia. To test it, the patient is placed on a line and made to move along it sideways in a given definite direction—say, towards the right. A normal individual during this movement, first leans his trunk to the left, then balancing his weight on the left leg, he lifts the right from the ground, abducts it by a muscular effort, brings the trunk erect again, puts down the right foot, last of all lifting the left leg, adducting it and placing it alongside the right. How is this performed in organic hemiplegia? We find that the hemiplegic patient moves sideways towards the paralysed side well, but badly towards the healthy side, so that in right-sided hemiplegia the patient, when going sideways towards the right moves normally, but when going towards the left he drags his right leg in the movement of adduction. This difference in the side-gait on the two sides in organic hemiplegia is often quite evident when the forward gait shows very little abnormality. To show the phenomenon properly, the patient must not be too severely paralysed to stand or walk, nor must he have shortening of the limb, as in old infantile hemiplegia. These two exceptions, however, are readily recognised by other signs. In hysterical hemiplegia the side-gait is impaired on both sides, not merely on the healthy side.

An *ataxic gait* is seen most typically in tabes dorsalis; but it occurs also to a lesser degree in other diseases implicating the posterior columns of the cord—for example, in Friedreich's ataxia, in chronic meningo-myelitis, or in tumours of the posterior columns, also in ataxic paraplegia or postero-lateral sclerosis, whether due to disseminated sclerosis or to other causes. Of all these, the tabetic gait is the most characteristic. In this there is no motor weakness of the limbs, but they are characteristically unsteady. The patient walks on too broad a base, with his legs unduly wide apart. He lifts them suddenly and violently, raising them too high, then bangs them down forcibly, stamping the heels on the ground. He tries to guide his tottering course by watching the ground. And therefore if his eyes be shut, or if he be in the dark, and especially if he narrows his base by placing the feet close together, he tends to fall. In slight cases of tabetic ataxia, when the patient is in this position, he may not actually fall, but we can see the tendons on the dorsum of the feet actively in movement, in the effort to preserve the balance. With this gait we usually have absence of the knee and ankle-jerks, Argyll-Robertson pupils, and various other signs, such as lightning pains, anæsthesiæ, crises, and lymphocytosis of the cerebro-spinal fluid. In *Friedreich's ataxia* the patient is usually an adolescent, and though the knee and ankle-jerks are absent, as in tabes, the pupils react normally, there are no lightning pains nor crises, and we usually find nystagmus, scoliosis, pes-cavus, and a peculiar affection of articulation. In *ataxic paraplegia* or *postero-lateral sclerosis*, where the lateral columns are implicated as well as the posterior, the patient is ataxic, but with increased knee-jerks, possibly ankle-clonus, and usually an extensor type of plantar reflex.

A *reeling* or *titubating gait* is one of the commonest signs of *cerebellar disease*, although it is also met with in severe vertigo of any variety, whether from alcoholic intoxication, from ear disease, from diplopia, or other cause. In cerebellar disease the patient staggers along, with an occasional sudden lurch to one or other side, but neither lifting his feet too high nor stamping them down, as in tabes. Not uncommonly the cerebellar patient has a tendency to stagger persistently in some particular direction, depending on the position of the cerebellar lesion—thus he may tend to fall forwards, backwards, or to one side. As a rule, he manages to pull himself up after he has deviated one or two steps from his straight course. In some cases of unilateral tumour of the cerebellum the posture of the head is altered, the ear being tilted towards the shoulder on the side opposite to the lesion, and the face turned slightly in the reverse direction, *i.e.* towards the side of the lesion. This is well seen in Fig. 117, which represents a woman who had a tumour originating from the meninges over the right petrous bone, and invading the right lateral lobe of the cerebellum. This growth was successfully localised and removed, but the patient unfortunately died shortly after. This "cerebellar attitude," however, although common in lateral lobe tumours, is not constant in its direction. For example, cases have been recorded where the lateral tilting of the head was towards the side of the lesion, and the rotation of the face to the opposite side. Possibly these differences are associated with the intra- or extra-cerebellar origin of the growth.

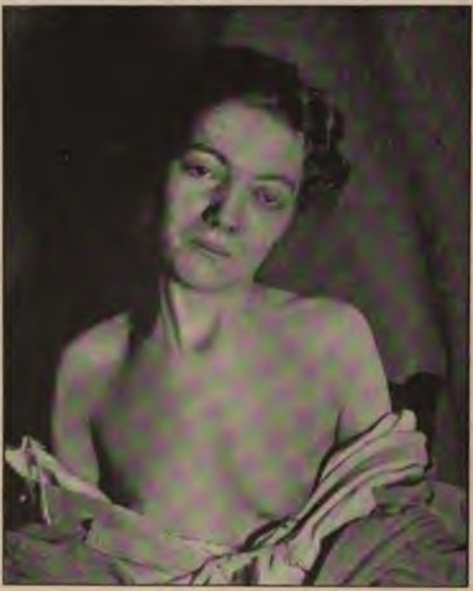

FIG. 117.—Case of right-sided cerebellar tumour arising from meningeal sheath of auditory nerve.

A *high-stepping gait* occurs in patients who have foot-drop. Such a patient, to clear his foot from the ground, lifts the leg too high, flinging the ankle up as it were, instead of actively dorsiflexing it. This gait occurs typically in *peripheral neuritis*, also in *muscular dystrophies*, and occasionally in lesions of the *cauda equina* or lower part of the *lumbo-sacral region of the spinal cord*. It also occurs unilaterally in paralysis of the *external popliteal nerve*. The gait and posture of muscular dystrophy also possess other characteristic features. Thus from weakness of the gluteal muscles the patient in the erect posture arches his back, in order to keep the hips extended (see Fig. 118); this produces lordosis and "pot-belly." Further, the weakness of the glutei, as already explained, causes a waddling gait, the legs being planted wide apart; and as we have already seen, the mode of rising from the ground in such cases is pathognomonic. Such a patient when placed on the ground and told to get up rolls round on his face, then gets on his hands and knees. Then to get on his feet, he extends the knees, and suddenly, pressing his hands on one knee after the other, proceeds to extend the hips and straighten the spine by "climbing up" his own thighs until he reaches the erect posture.

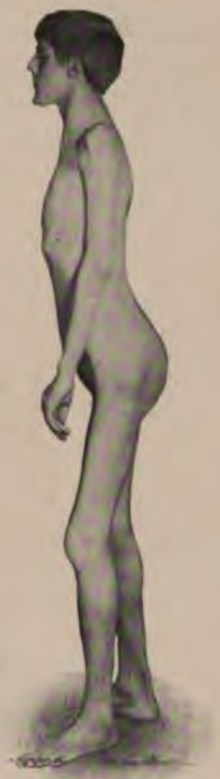

FIG. 118.—Muscular dystrophy in a lad of 17. Showing lordosis.

The posture and gait of *paralysis agitans* are diagnostic (see Figs. 119 and 120). In a well-marked case the patient stands with the trunk stooping forwards, the face appearing "starched" and expressionless—the so-called "Parkinsonian mask," in which there is little or no emotional play of features. The upper limbs are slightly abducted at the shoulders, semi-flexed at the elbows, slightly extended at the wrists, flexed at the metacarpo-phalangeal joints, and extended at the inter-phalangeal joints, as if holding a pen—the "interosseal" attitude—and very often they show the familiar rhythmic, "pill-rolling" tremor. The tremor may affect the proximal joints as well, and even the lower limbs, face, jaw, palate, and tongue. All the voluntary movements of the trunk and limbs are slow and stiff, and the gait is "festinant." The patient moves forward with short, shuffling steps, and when he turns, his trunk moves slowly *en masse*, as if made of glass, whilst his steps in walking tend to get faster and faster, as if he were "chasing his own centre of gravity." This is called "propulsion." Still more frequently do we observe "retropulsion," in which the patient, when pulled gently from behind, tends to run backwards with short, hasty steps. This retropulsion may sometimes be induced even by the act of looking upwards. A slighter degree of this same posture and gait is not uncommon in simple old age.

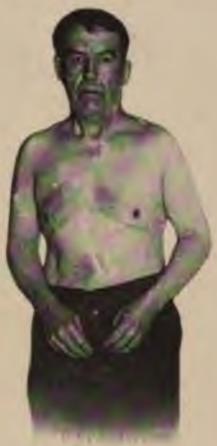

FIG. 119.—Paralysis agitans—bilateral.

The gait in *chorea* is sometimes peculiar, partly owing to a degree of ataxia, partly from the presence of additional irregular involuntary movements. Sometimes one foot seems as if it were momentarily entangled by an invisible obstacle, which holds the child back for an instant, the patient then hastily resumes his forward progress; or his knee may give way suddenly, causing him to fall.

Many chronic *epileptics* have a peculiar slouching posture and gait, the posture of the hands, as Spratling[1] has pointed out, being specially characteristic. The fingers are habitually flexed, and the wrists bent to a right angle, and the patient often has a marked forward stoop of the shoulders.

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[1] *New York Medical Journal*, 1905, p. 849.

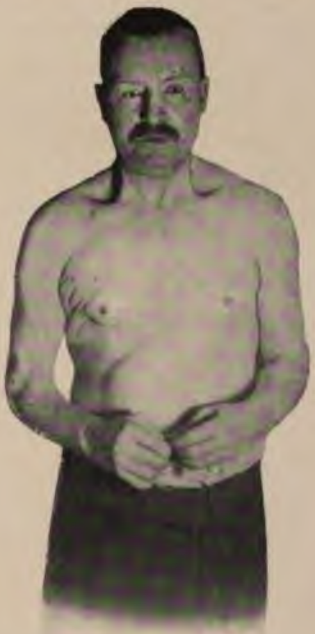

FIG. 120.—Paralysis agitans—bilateral.

Various *deformities* produce abnormalities of gait and posture. If, for example, one lower limb is shortened from disease of the bones or joints, from congenital malformations such as coxa vara, or congenital dislocation of hip, or from infantile paralysis, the gait becomes correspondingly altered.

LECTURE XVIII

TROPHO-NEUROSES

THE central nervous system exercises a profound influence on the nutrition of all the tissues. There is, however, no evidence of the existence of special nerves whose function is trophic and trophic only. The control over nutrition which the nervous system possesses is probably exercised in a complex fashion, in which there are several factors involved. Striated muscles, for example, undergo atrophy when the cells of the corresponding motor nucleus in the cord or medulla are destroyed, or when the motor nerve-fibre leading from the nerve-cell to the muscle-fibre is interrupted. The result is muscular atrophy, the different varieties of which we have already studied (see p. 186). Afferent nerve-fibres conveying sensory impressions, whether conscious or subconscious, have also a profound influence upon tissue-nutrition, especially upon that of the skin and its appendages. And therefore in anæsthetic areas trivial injuries are liable to produce destructive tissue-changes. Lastly, the central nervous system indirectly influences the tissues through their blood-supply, by means of its connections with the sympathetic vasomotor system. The sympathetic system may also be disordered primarily, apart from the central nervous system, not only in gross lesions of the sympathetic chain, but also in the so-called angio-neuroses.

Excluding, then, the muscular atrophies and the angio-neuroses, which are studied elsewhere, let us direct our attention to certain trophic disorders which are associated more or less directly with affections of the cerebro-spinal nervous system. Trophic disorders may be distributed widely all over the body, or they may be limited to certain circumscribed areas corresponding to a peripheral nerve, to a posterior root, or to some division of the spinal cord or brain.

Generalised Trophic Disorders.—Of these, one of the best examples is the well-known *anorexia nervosa*. In this affection, without evidence of structural disease of any organ, the patient (generally a young woman) loses appetite and becomes progressively emaciated, often to a profound degree. The condition sometimes follows a shock, physical or mental, perhaps an *affaire du cœur*, though in other instances we can find no apparent exciting cause. In diagnosing this condition, we have first to exclude other conditions, such as diabetes, tuberculosis, and malignant disease, which commonly produce emaciation. The patient generally exhibits certain "stigmata" of functional disease. Of these stigmata the most frequent is a hemianæsthesia, usually slight in degree and generally left-sided (see later, p. 314).

In marked contrast to this is the rare affection known as *adiposis dolorosa*, or Dercum's disease. It occurs chiefly in middle-aged women, many of the patients being alcoholic or syphilitic. The patient is diffusely obese, and, in addition, she has localised fatty lumps in the subcutaneous tissue, forming large pendulous swellings, chiefly on the limbs and trunk. These swellings may be symmetrical or asymmetrical. They appear and steadily increase in size, consisting of fat and of an embryonic form of connective tissue. The arms are most frequently the sites of the swellings, which are usually tender on pressure and may have spontaneous pains. Certain areas, however, such as the hands, feet, and face, are always spared. The nerve-trunks are tender, and there may be areas of blunting or loss of cutaneous sensibility. Sometimes the thyroid gland is indurated, but the patient has none of the mental or physical features of myxœdema.

A curious condition of the bones results from hypertrophy or functional over-activity of the pituitary gland.[1] Hyper-secretion by this gland appears to set free in the body certain abnormal substances whose action is to cause an extraordinary growth of bony tissues. If the disease sets in before the age at which the epiphyses have become joined, the bones grow enormously in all their dimensions. and the result is *gigantism*. But if the affection begins after the epiphyses have united, the overgrowth of the bones is confined to their ends, producing acromegaly.

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[1] Acromegaly cannot be ascribed, as was formerly thought, to deficient pituitary secretion, for as Tamburini and Modena have pointed out (*Rivista sperimentale di Freniatria*, 1903, fasc. 3 and 4), experimental destruction of the gland does not cause acromegaly, nor do malignant growths, nor tuberculous disease of the gland produce it, but only conditions such as hypertrophy or adenoma.

The phenomena of *acromegaly* are very characteristic. There is a progressive enlargement of the bones and soft parts, most marked in the hands and feet, but also affecting other parts, notably the skull and face. The skull becomes enlarged and thickened, all its bony ridges are exaggerated; the margins of the orbits, the cheek-bones, and most striking of all, the lower jaw, become enlarged. The mandible becomes prognathous, the lower teeth biting in front of the upper, instead of *vice versâ*, and the teeth may become widely separated. The soft parts also share in the hypertrophy. The lower lip, the tongue, uvula, tonsils, and the cartilages of the ears, all become enlarged, and the skin of the face becomes thick and coarse. The hands and feet increase in size, so that the patient requires gloves and shoes several sizes larger than before. Spinal curvature is also present, usually a cervical kyphosis, and the thorax, pelvis, and even the external genitals become enlarged. Glycosuria is a fairly common complication. In female patients amenorrhœa occurs. In addition to these phenomena the patient complains of severe headaches, owing to the intra-cranial pituitary tumour. This tumour, from its position in the *sella turcica*, frequently encroaches on the adjacent optic chiasma, and then there is produced a corresponding affection of the visual fields (see p. 101), usually commencing as a bi-temporal hemianopia, which may, as the disease advances, progress to complete blindness.

Apart, however, from pituitary disease which strictly speaking, although an intra-cranial disease, is not primarily a nervous disorder, we sometimes have widespread overgrowth of the tissues, confined to one-half of the body and probably of cerebral origin Fig. 121 represents such a patient with left-sided *hemi-hypertrophy*, in whom all the bones of the left side (as verified by skiagrams), including those of the limbs, pelvis and thorax, together with the soft tissues such as the face and tongue, were larger than on the right side. But the right side of the cranium, and probably also the right side of the brain, was larger than the left.

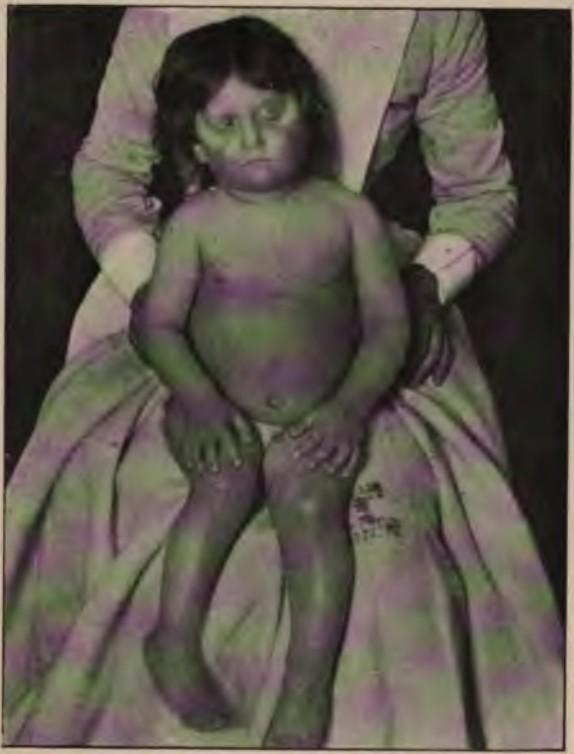

FIG. 121.—Left-sided hemi-hypertrophy.

Passing next to trophic disorders of more limited distribution, it is convenient to discuss them in certain groups.

Trophic Changes in the Skin and its Appendages.—*Glossy skin* is a condition met with chiefly in the hands, in certain cases of long-standing peripheral nerve palsies, whether traumatic or neuritic in origin. It is also a frequent accompaniment of osteo-arthritis, not only in the ordinary "rheumatoid" variety, but also in the arthritis which comes on in hemiplegic limbs. The skin of the fingers becomes thinned and atrophic, with a peculiar smooth, shiny surface. The nails in many cases are altered, being longitudinally striated and excessively curved from side to side. The finger-tips taper to a point, as is seen in Fig. 122, which is taken from a case of right-sided brachial neuritis.

Perforating ulcers occur most typically in tabes dorsalis. They are generally situated on the foot, on its plantar surface, especially at the metatarso-phalangeal joint of the great or little toe. They may also occur under the heel or under the terminal phalanx of the hallux. Each ulcer begins as a thickening of the epidermis, like a corn. Suppuration occurs under this, and the pus finds its way out through a small opening in the centre (Fig. 123). A narrow sinus is thus formed which increases in depth until it may extend into the joint beneath, which is often disorganised, and carious bone may be felt at the bottom. Sometimes the ulcer heals up under treatment. The tabetic perforating ulcer is painless. Somewhat similar trophic ulcers are met with in certain cases of *spina bifida occulta* and also in *syringomyelia*, but in this latter disease they are commoner in the hands. *Diabetic neuritis* is also occasionally associated with perforating ulcers of the feet,

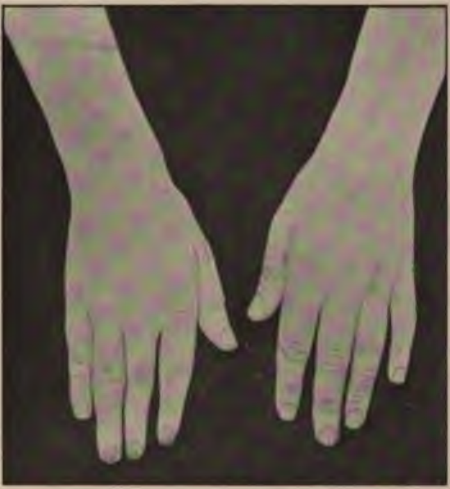

FIG. 122.—Right-sided brachial neuritis with glossy skin and tapering finger-tips.

a minor variety of diabetic gangrene. And in *leprous neuritis* perforating ulcers are not infrequent, though it is commoner to have a still more extensive loss of tissue, whole phalanges dropping from the fingers and toes. *Painless whitlows*—in so-called "Morvan's disease," a sub-variety of syringomyelia, are found at the finger-tips. These whitlows are sometimes the result of trivial injuries which in a normal individual would not produce any serious results. In other cases the explanation is found in the absence of sensibility to temperature and pain, which is characteristic of the disease, so that the patient burns the fingers painlessly and produces blisters and even areas of sloughing.

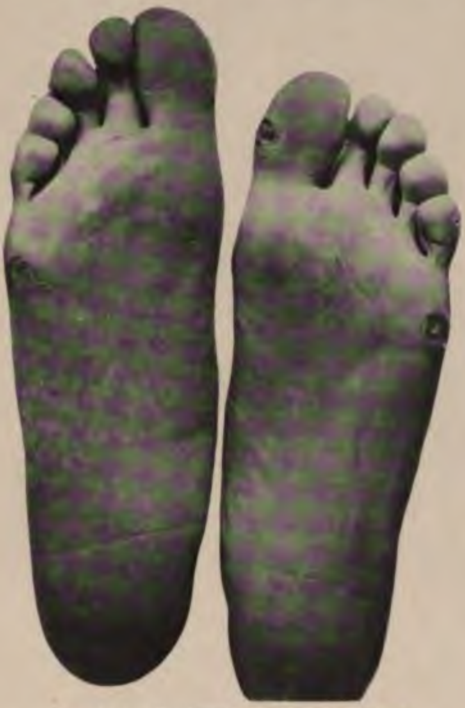

FIG. 123.—Perforating ulcers in tabes.

In certain cases of tabes there may be a perforating ulcer in the mouth. First the teeth become loosened and fall out, then the alveolar margin of the jaw is absorbed, and if the upper jaw be affected a perforation into the nasal cavity may be established.

Herpes zoster is a very typical example of a trophic cutaneous disorder which has a direct nervous origin, viz., inflammation or thrombosis of the corresponding posterior root ganglion. In this affection a crop of vesicles appear, which are distributed in a definite area, corresponding to the posterior root whose ganglion is diseased. In the case of facial herpes it is the Gasserian ganglion which is inflamed, in whole or in part. The herpetic vesicles usually become pustular and subsequently permanent scars may remain. Herpes is sometimes preceded, for a day or two, by pre-herpetic pain in the area where the eruption is about to appear; and not infrequently the eruption is followed by prolonged and intractable post-herpetic neuralgia. Figs. 124 and 125 are from a typical case of herpes in the area of the fifth thoracic root. Sometimes herpes developes in the course of diseases of the

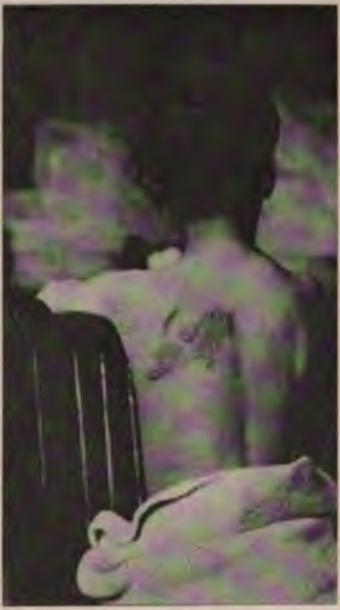

FIG. 124.

Herpes zoster in area of fifth dorsal root. Second day after appearance of eruption.

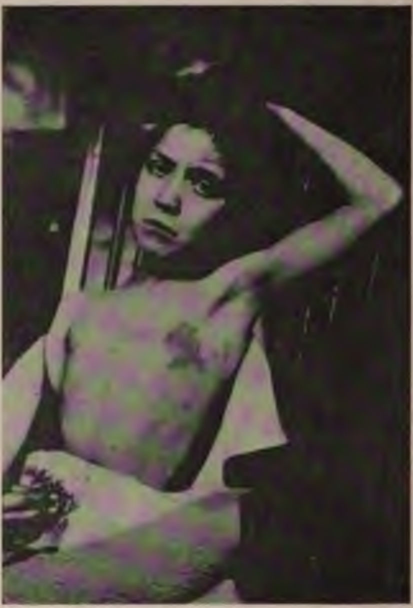

FIG. 125.

Herpes zoster—the same patient as in Fig. 124. Eruption surrounds the nipple, and thence extends inwards to mid-sternum.

vertebræ or of the spinal meninges. Such an occurrence signifies that the morbid process has attacked the corresponding posterior root ganglion or ganglia.

Bed-sores result from inflammatory and destructive changes in the skin and underlying tissues of bedridden patients, whether in cases merely enfeebled by prolonged illness (especially in enteric fever) or, more commonly, in severe organic paralysis, such as hemiplegia or paraplegia. Most bed-sores, occurring as they do at the sites of pressure, can be prevented by careful nursing, by keeping the patient's skin scrupulously clean and dry, by placing him on a water-bed, and by hardening the epidermis by local applications of methylated spirit. But sometimes in spite of the most assiduous nursing, bed-sores may develope within a few days or even within a few hours of an initial paraplegia or hemiplegia. This so-called *acute decubitus* is of grave omen.

The commonest site for a bed-sore in a hemiplegic patient is over the great trochanter on the paralysed side. In paraplegia from cord lesions, *e.g.* acute myelitis, the bed-sore commonly forms over the middle of the sacrum (Fig. 126). A bed-sore commences as an area of redness of the skin, bullæ then develope and burst, leaving an ulcerating or sloughing surface beneath. The sloughing process may extend down to the periosteum and bones, and in sacral decubitus the infection may extend into the vertebral canal, producing a fatal cerebro-spinal meningitis, septic organisms gaining access not only from the skin, but from the intestinal discharges. Bed-sores are also met with in advanced cases of bedridden dementia, even where there is no paralysis. In these patients, as in the bed-sores of prolonged fever, the sores develope on the pressure points, viz. the heels, hips, backs of the scapulæ and even on the inner sides of the knees. We also meet with excoriations of the skin, which may amount to bed-sores, in cases of violent chorea, where the patient knocks his limbs against surrounding objects.

Before leaving the subject of destructive trophic lesions of the skin, we must mention *symmetrical gangrene*, a localised gangrene chiefly occurring in the tips of the fingers and toes, and preceded by pains or paræsthesiæ of the parts. But to this, the most severe phase of Raynaud's disease, we shall refer again when we consider the angio-neuroses.

The commonest points of incidence of *rodent ulcers*, especially on the face, as Cheatle[1] has shown, are at the positions where the various branches of the trigeminal nerve becomes cutaneous, especially over the points of emergence of the infra-orbital, infratrochlear, temporo-malar and lachrymal nerves, and also with

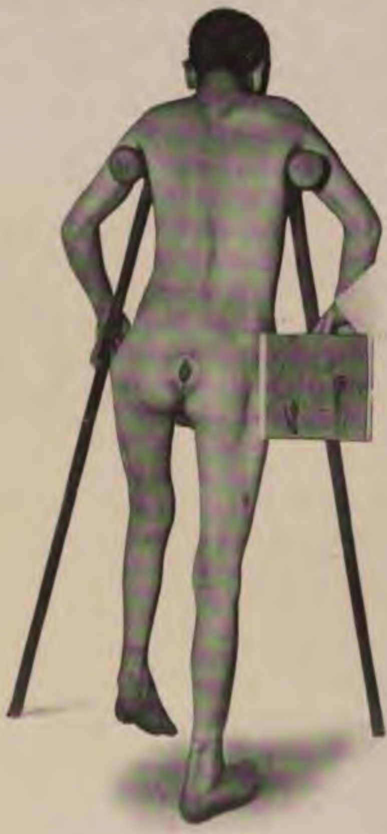

FIG. 126.—Lumbo-sacral myelitis with sacral bed-sore or decubitus. Showing atrophy and paralysis of muscles below left knee. The small chart on the right indicates the coexisting area of anæsthesia.

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[1] *Brit. Med. Journal*, April 29, 1905.

particular frequency at the inner canthus of the eye and at the angle where the ala nasi joins the cheek. He has also shown that these spots are the favourite points of incidence of leucoderma and scleroderma. The precise significance of these facts is still obscure, but Cheatle has recorded the curious fact that rodent ulcers are frequently limited to the distribution of normal nerve-areas and that the spread of a rodent ulcer ceases abruptly when it reaches a cutaneous area which has become denervated by division of its sensory nerves.

There are other trophic lesions unaccompanied by necrosis or ulceration, and consisting in local changes affecting one or more elements of the skin or subcutaneous tissues, whether in the direction of atrophy or of hypertrophy. Perhaps the most typical examples of this variety are scleroderma and leucoderma. *Scleroderma* is a disease in which the skin becomes abnormally hard and fibrous. The condition may be diffuse or circumscribed. In the rarer diffuse variety, large areas of skin, usually symmetrical, and especially in the upper limbs, become hard and rigid, losing their elasticity so that the affected skin can no longer be pinched up with the fingers. Sometimes there is a preliminary œdematous stage. As the disease advances, the sclerotic process may extend to deeper structures such as tendons, and this, with the rigidity of the skin, limits the movements of the joints and may produce permanent deformities. If the fingers are affected, they become tapering at the tips and permanently flexed. If the face is affected, it becomes immobile and mask-like. Circumscribed scleroderma, or morphœa, is the commoner variety, where small patches of skin become hard, white and ivory-like, the distribution of the patches being somewhat similar to those of herpes zoster—*i.e.* in the area of a posterior root, or of one of the divisions of the trigeminal nerve on the face.

Leucoderma, or disappearance of pigment from circumscribed patches of skin, is also commonly distributed, more or less, in nerve-areas. The patch of absolute pallor has a pigmented edge where it joins the normal skin, and it tends to spread slowly along the particular area. This disease, of course, is most striking when it affects patients of sallow complexion or of dark-skinned race. Leucoderma also occurs in the maculo-anæsthetic type of *leprosy*, where the leucodermic patches are often red and hyperæsthetic at the edges and anæsthetic in the centre.

Various cutaneous lesions are present in many cases of *arsenical neuritis*. A brownish macular pigmentation of the skin is, of course, common in chronic arsenical poisoning, without neuritis. But in arsenical neuritis we frequently observe special cutaneous affections, such as glossy skin, herpes zoster, falling of the hair, and most characteristic of all, hyperkeratosis of the palms and soles, where the epidermis becomes much thickened and tends to desquamate. In addition to these skin lesions, we have the ordinary signs of a peripheral neuritis, such as drop-wrist, drop-foot, &c.

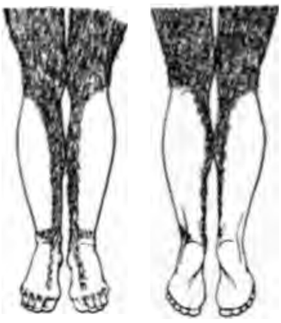

FIG. 127.—Case of tabes with loss of hair in area of L_5 and S_1 roots.

There is a rare trophic affection of the skin, described by Gowers as *local panatrophy*, in which certain circumscribed areas of the face, trunk, or limbs, varying in size from the diameter of a cherry to that of an orange, undergo local atrophy of all the subcutaneous tissues down to the bone, the skin becoming also slightly thinned. These patches look like subcutaneous excavations, and although trophic in origin, they do not correspond to regular nerve-areas but are quite irregularly distributed.

Trophic changes are sometimes present in the hair. *Hypertrichosis*, or excessive growth of the hair, is met with most frequently on "hairy moles," which are, moreover, excessively pigmented in the skin as well. It is important to remember that a hairy mole in the lumbo-sacral region is often an indication of a *spina bifida occulta*. Local hypertrichosis also occurs occasionally in other nerve-areas. The hair may also be affected in various ways in other nervous affections. Well-authenticated cases have been recorded of *blanching of the hair* of the scalp within a few hours, as a result of profound emotion. It is not uncommon to meet with patches of whitened hair in nerve-areas which have been the site of severe neuralgia. Even in the ordinary greyness of advancing years, and in the idiopathic premature greyness of youth, as Cheatle[1] has pointed out, the maximum greyness often appears in nerve-areas in the scalp and beard. There is

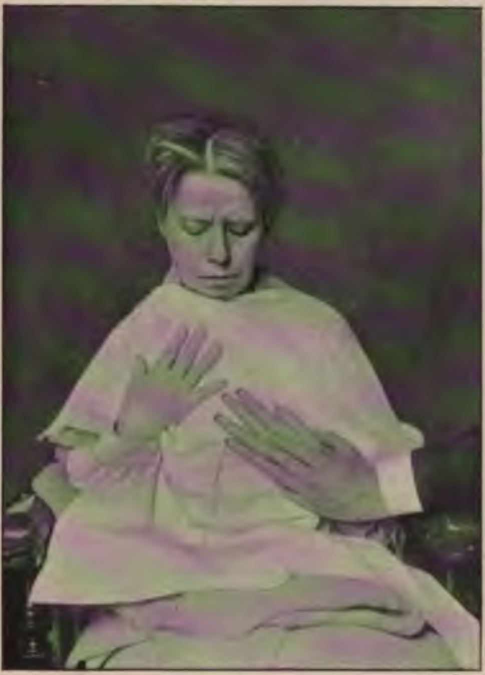

FIG. 128.—Case of left-sided hemiplegia with hæmorrhages under all the finger-nails on the left side, and under the nail of the right little finger.

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[1] *Brit. Med. Journal*, July 22, 1905.

a variety of *localised alopecia* where the hair falls out suddenly in a certain nerve area. Fig. 127 is from a case of tabes where there was a curious absence of hair in certain root-areas in the legs. We occasionally meet with cases of *universal alopecia*, where the hairs all over the body fall out, and the patient may remain permanently hairless, his skin meanwhile being considerably thinned.

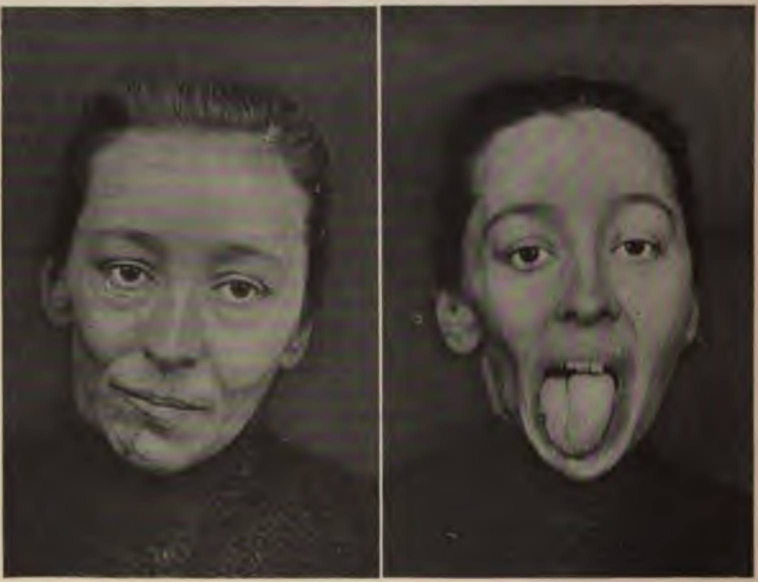


FIG. 129 FIG. 130.

Right-sided facial hemiatrophy, also implicating the corresponding side of the tongue.

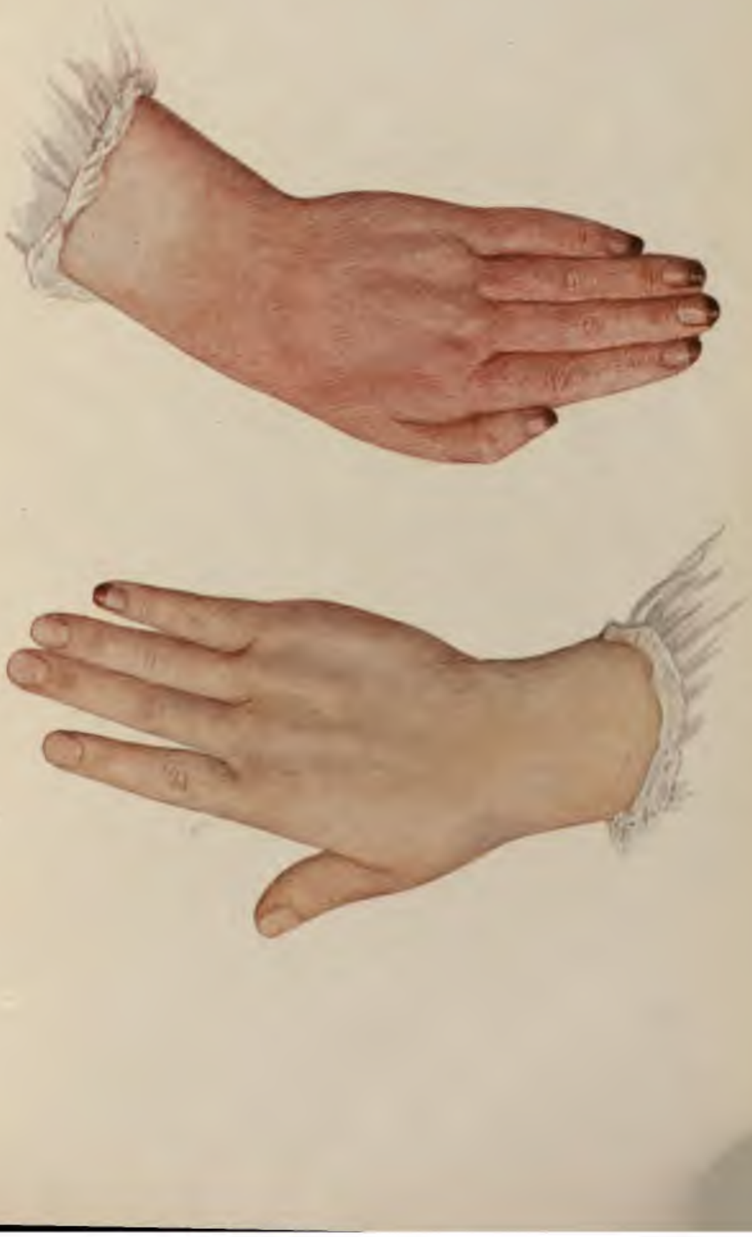
Trophic changes in the *nails* occur in a number of nervous diseases. Sometimes the nails become hypertrophied, as in *acromegaly* and in the toe-nails of patients with *chronic paraplegia* from any cause. In *peripheral neuritis*, especially the arsenical variety, we may meet with excessive curving, brittleness, atrophy and even falling of the nails. In *tabes* the toe-nails, especially those of the big toes, are sometimes shed, whilst in some cases of *cerebral hæmorrhage* we find hæmorrhages under the nails of the hemi-

PLATE I.

Sub-ungual hæmorrhages in a woman aged 42 in whom, eighteen months after an attack of left hemiplegia, there occurred acute swelling and redness of the left hand, to a lesser degree in the left foot, and, a few days later, in the right little finger. The affected finger-tips became deeply cyanosed, bullæ developed on the affected hand, and were followed by desquamation.

The drawing shows the condition two and a half months later, the nails having grown to some extent. The left hand was hotter than the right, and perspired more freely.

To face page 258.



plegic hand (Fig. 128 and Plate I.). As the nail grows, the hæmorrhagic area is gradually cast off.

Trophic Changes in Bones and Joints.—An affection which possesses characters common to this group and to the group of cutaneous trophic lesions is *progressive facial hemiatrophy*. It is probably referable, as we have already seen, to a lesion of the trigeminal nerve or nucleus. Not only is the skin on the affected side of the face atrophied and wrinkled, but the bones, and especially the lower jaw, become smaller (Figs. 129 and 130). Sometimes, on the other hand, we meet with cases of *facial hemi-hypertrophy*, where the bones and soft parts of one side of the face become progressively larger. In the case shown in Fig. 131, the enlargement of the face followed an injury to the forehead in childhood on the side which afterwards became enlarged.

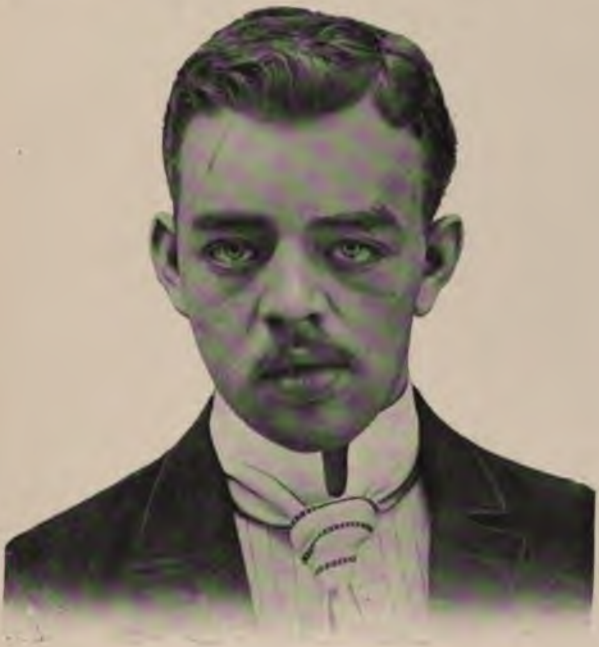


FIG. 131.—Hemi-hypertrophy of right side of face.

It is not uncommon to find atrophic changes in the bones of paralysed limbs. In paralytic affections of children, whether of the upper neurone type, as in *infantile hemiplegia* (Fig. 132), or of the lower neurone type, as in *acute anterior poliomyelitis* (Fig. 133), the bones of the paralysed limb become arrested in their growth and are smaller in all their dimensions than in the healthy limbs. And even in some cases of *hemiplegia in adults* the bones of the paralysed limbs become excessively brittle. I have known a case of hemiplegia in a middle-aged woman where moderate passive movements under an anæsthetic, during an attempt to break down adhesions in the hip-joint of the hemiplegic side, caused a fracture of the neck of the femur. The brittleness of the bones in certain

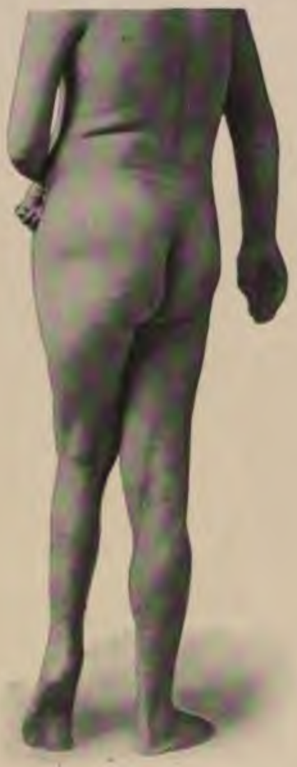


FIG. 132.—Old infantile hemiplegia, left-sided, with arrested growth of limbs.

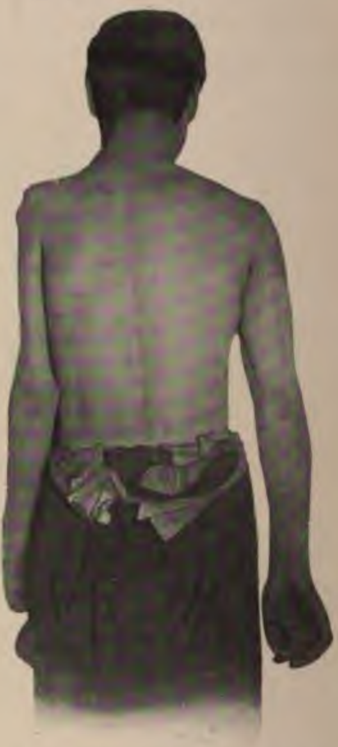


FIG. 133.—Old anterior poliomyelitis anterior acute. Paralysis and atrophy of biceps, triceps, and deltoid on left side, with arrested growth of humerus.

insane patients and their liability to fractures on trivial injuries is well known. And there is a rare disease known as idiopathic *fragilitas ossium*, where the patient, otherwise healthy, may fracture his bones from minimal accidents, as, for example, where a patient fractured his humerus by throwing a cricket-ball.

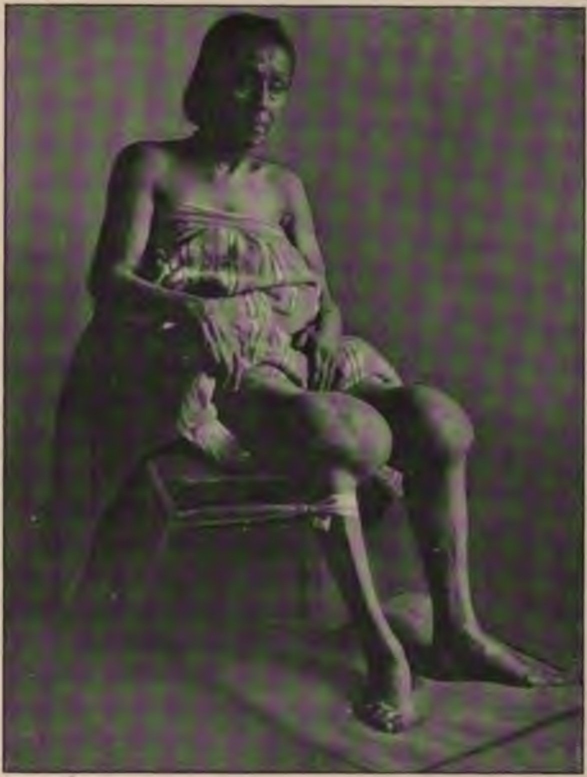


FIG. 134.—Tabetic arthropathy of both knees and of right foot.

Similar spontaneous fractures also occur in certain cases of *tabes* and of *syringomyelia*, but in these two diseases the fractures are painless, so that the patient may continue to use the fractured limb in a fashion impossible to a normal individual.

Trophic changes in joints—the so-called *arthropathies*, are met with most typically in tabes (constituting Charcot's joint), in syringomyelia, and in some cases of leprosy. The large joints are generally those affected, the knee and the tarso-metatarsal joint being the joints most commonly attacked in tabes (Fig. 134), the shoulder in syringomyelia (Figs. 135 to 138). But in tabes even the smaller joints may occasionally be affected, as in the patient shown in Fig. 139, where the terminal joint of the thumb was thus diseased. Female tabetics seem to be relatively more liable than male patients to osteopathies and arthropathies. The exciting cause of a tabetic arthropathy is often some trivial traumatism, such as a twist or sprain in a joint in which the sense of pain is diminished or lost. Hence the greater frequency of tabetic arthropathies in the lower limbs. The joint swells painlessly, and rapid destructive changes occur in its articular surfaces, which become eroded and may disappear, together with a considerable part of

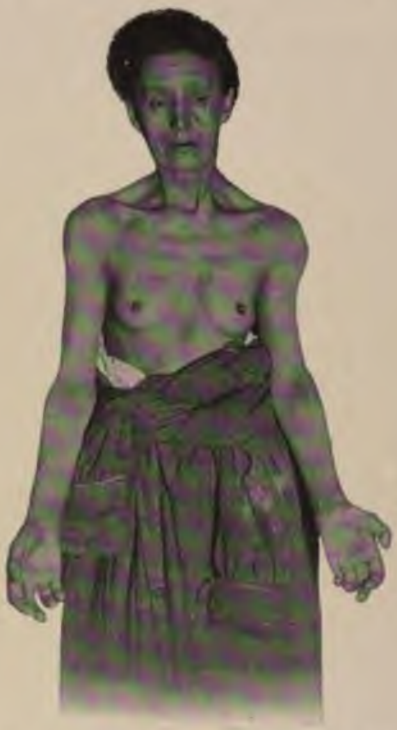


FIG. 135.—Syringomyelia with arthropathy of left shoulder-joint. Atrophy of intrinsic muscles of left hand.

the adjacent bone. Fractures of the articular ends are common, both in tabetic and in syringomyelic joints. The ligaments become lax and the joint abnormally mobile, so that in the knee we may be able to produce lateral passive movements, or even to bend the joint into all sorts of curious positions (Fig. 140). The joint is sometimes distended with a glairy gelatinous fluid, which may be blood-stained. In the later stages this fluid may be reabsorbed, throwing into relief the deformity of the bones (Fig. 141). But the changes in such arthropathies are not entirely destructive. Osteophytic outgrowths are often formed in the peri-articular tissues, causing irregular thickening of the

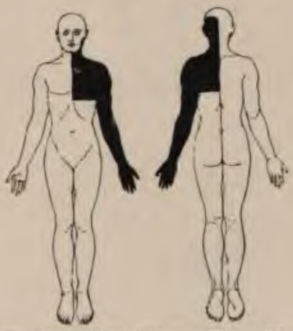


FIG. 136.—The same case of syringomyelia. Showing the area of therm-anæsthesia and analgesia.

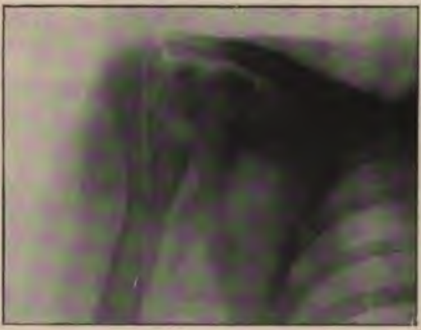


FIG. 137.—Syringomyelia (same case as Fig. 135). X-ray photograph of shoulder-joint. Showing fracture of upper end of humerus, also osteophytic outgrowth from axillary border of scapula.

bones, and producing little islands of new bone amongst the tissues around the joint. These can be readily detected on palpation and verified by skiagrams (Figs. 142 and 143).

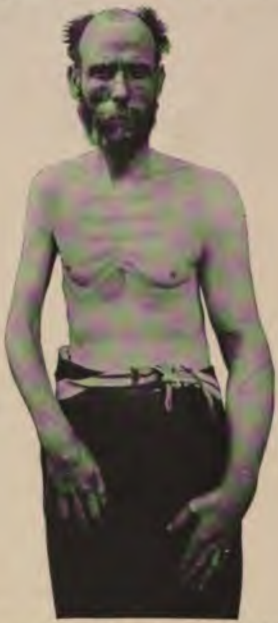


FIG. 138.—Syringomyelia with arthropathy of left shoulder-joint.

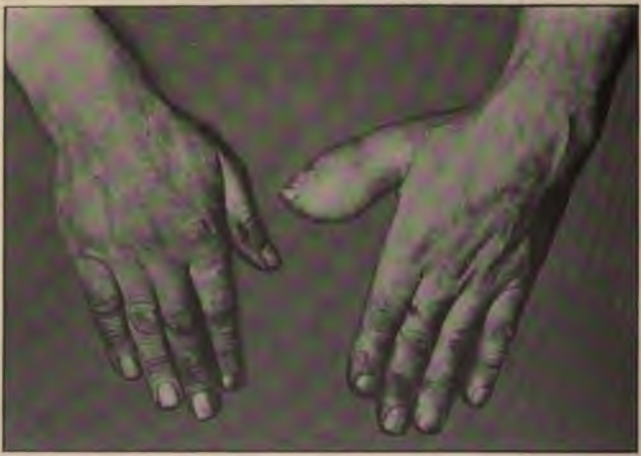


FIG. 139.—Tabetic arthropathy of left thumb.

Joint affections of a different kind are met with in some cases of chronic hemiplegia. The joints of the paralysed limbs, two or three weeks after the hemiplegic attack, become swollen and deformed. But, unlike the tabetic and syringomyelic arthropathies, hemiplegic joint-affections are acutely painful. Moreover, the changes in hemiplegic joints are not destructive but more of the nature of a subacute osteo-arthritis, with pain and rigidity on passive movements and with a tendency to the formation of fibrous adhesions within the joint and to thickening of the joint capsule. When the fingers are affected, they lose their normal outline and become clumsy and "sausagy" in appearance. Together with this hemiplegic arthritis it is not uncommon to have a degree of muscular wasting, but without electrical reactions of degeneration.

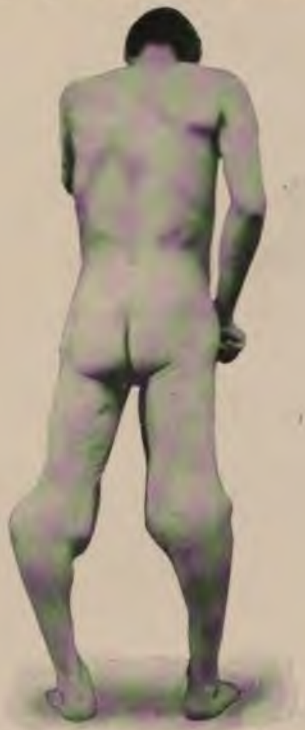


FIG. 140.—Tabetic arthropathy of knee-joints.

Finally, we must bear in mind that certain *hysterical* patients complain of joint-pains, usually mono-articular, which may more or less closely simulate organic joint disease. The patient is commonly a young woman who, after some local injury to the joint, or after some emotion, or perhaps from being acquainted

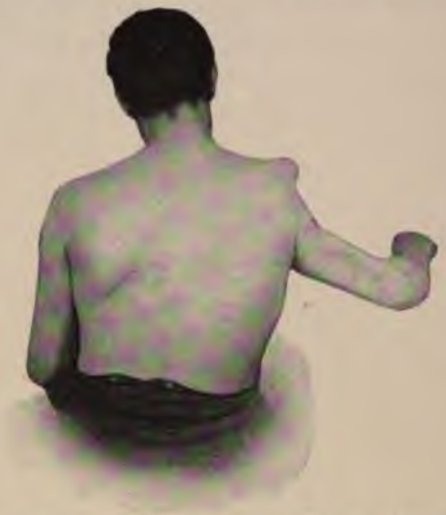


FIG. 141.—Syringomyelia with arthropathy of right shoulder-joint, and destruction of head of humerus. Well-marked scoliosis.

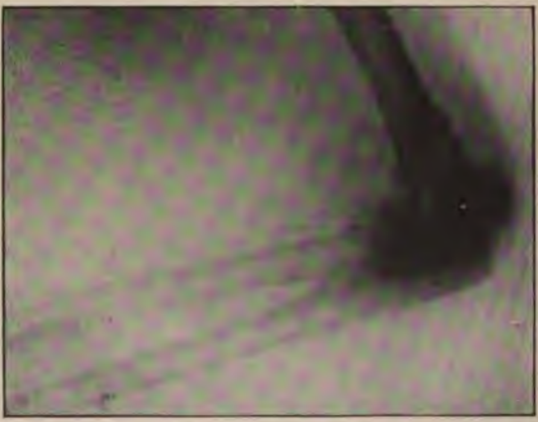


FIG. 142.—Tabetic arthropathy of elbow-joint. X-ray photograph, showing bony deposits in the tissues around the joint.

with another patient who has joint disease, suddenly complains of intense joint-pain, together with cutaneous hyperæsthesia in that region. The joint is rigid and resistant to passive movement, and if the condition has persisted, as is sometimes the case, for weeks or months, fibrous adhesions may form. But there is no true swelling of the joint when compared with the corresponding limb of the opposite side, though a false appearance of swelling may appear to be present owing to disuse-atrophy of the adjacent muscles. The presence of other hysterical "stigmata" often aids in the diagnosis, but an accurate opinion is sometimes a matter of considerable difficulty and is arrived at by a process of exclusion. Accomplished surgeons have been known to operate on such joints and only on opening the joint to have discovered the absence of organic disease. The result of such operations, fortunately, is that the patient usually recovers from her pains.

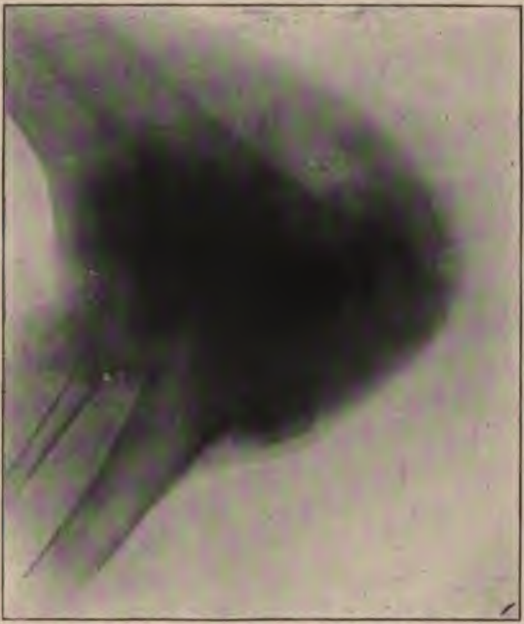


FIG. 143.—X-ray photograph of tabetic arthropathy of knee, from patient shown in Fig. 134. Showing disappearance of articular surfaces and presence of bony deposits in peri-articular tissues.

LECTURE XIX

REFLEXES

FOR clinical purposes we have to consider three varieties of reflexes :—

(1) Superficial or skin reflexes, *e.g.* the plantar reflex.

(2) Deep or tendon reflexes, *e.g.* the knee-jerk.

(3) Organic or visceral reflexes, *e.g.* vomiting, micturition, parturition.

Of these three groups, the first two have to do with striated voluntary muscles. They have their reflex centres within the cerebro-spinal axis, and many of them can be inhibited by voluntary effort. The third group, that of the visceral reflexes, is concerned not with voluntary muscles alone, but also with involuntary, non-striated muscles which are controlled by the sympathetic nervous system and are incapable of direct voluntary restraint—*e.g.* the muscular walls of the stomach, bladder, rectum, or uterus. Such visceral reflexes can be performed, more or less perfectly, independently of the central nervous system. Normally, visceral functions go on, for the most part, unconsciously. But even they may occasionally evoke consciousness, as when visceral pain occurs, or when striated voluntary muscles are required to supplement an act originally initiated by non-striated muscles, or when the reflex act causes stimulation of a cerebro-spinal sensory nerve. Thus, to take an example, the act of defæcation is practically unconscious and uncontrollable, so far as the movement of the large intestine is concerned. But when the lower end of the anal canal has its mucosa stimulated, the perineal muscles come into action, and the act then becomes a conscious one and is more or less under control.

Skin Reflexes.—These are movements obtained by slight stimulation of certain areas of skin or mucous membrane. The result is a movement, quick or slow, of the skin near the point of stimulation, but not exactly under it. In certain animals such as the horse, skin reflexes can be obtained by touching almost any part of the trunk. But in man the skin as a whole is less mobile, and it is usually only from certain special areas that skin reflexes can be elicited. If the stimulus be too strong, it may cause a reflex so violent as to involve almost all the voluntary muscles of the body. Or if the stimulus, even though slight, be a painful one, as for example a pin-prick, the result is a rapid "defensive" movement—*e.g.* sudden flexion of a limb or sudden closure of the eye.

The following table gives a list of the chief skin reflexes which are of clinical importance, and the mode of testing each. All are more easily elicited in young people than in old; in fact, if we wish to demonstrate the skin reflexes with certainty, we select a child for our subject.

	METHOD OF ELICITING.	RESULT.	SEGMENTAL LEVEL.
Conjunctival	Touching cornea.	Orbicularis oculi contracts.	
Pharyngeal	Touching posterior wall of pharynx.	Pharynx contracts.	
Palatal	Touching soft palate.	Palate is elevated.	
Scapular	Stroking skin in inter-scapular region.	Scapular muscles contract.	C5 to D1.
Epigastric	Stroking downwards from nipple.	Epigastrium dimples on side of stimulus.	D7 to D9.
Abdominal	Stroking downward from costal margin.	Abdominal muscles contract on side of stimulus.	D11 to L1.
Cremasteric	Stroking inner and upper part of thigh. Or pressure over Hunter's canal.	Testicle is pulled up.	L1 and L2.
Gluteal	Stroking skin of buttock.	Gluteal muscles contract.	L4 and L5.
Plantar	Stroking sole of foot.	Tensor fasciæ femoris contracts, hallux and other toes flex, ankle is dorsi-flexed.	L5 to S2.
Bulbo-Cavernosus	Pinching dorsum of glans penis.	Bulbous urethra contracts.	S3 and S4.
Superficial Anal	Pricking skin of perineum.	External anal sphincter contracts.	S5 and Coccygeal.

Absence of the *abdominal reflex* is not uncommon in acute abdominal conditions, notably in appendicitis and enteric fever.[1] In young adults whose abdominal walls are apparently normal and in whom there is no œdema or excessive obesity, absence of this reflex is strongly suggestive of disseminated sclerosis, as has been pointed out by Strümpell, E. Müller,[2] and others.

Of all the superficial reflexes, the *plantar reflex* is the one which has the greatest practical importance. In order to test it, the patient should be lying down, his feet being comfortably warm. The limb to be tested is now partially flexed at the hip and knee, and also rotated so as to rest on its outer side. Then with some hard object such as the end of a penholder, we gently stroke the sole of the foot from behind forwards, especially towards its inner side. Meanwhile we watch carefully for the first movement of the great toe. Normally this is a movement of plantar flexion (see Fig. 144). The movement of the other toes is of less importance. It is interesting also to note that, simultaneously with the toe movement (a "cortical" reflex) we have a brisk contraction of the tensor fasciæ femoris, medullary in origin, which may occur even in cases where the toes show no movement. This proves that the plantar reflex is not a phenomenon confined to the foot but that it implicates the whole lower limb. If we employ a stimulus which is too strong, we may have, in addition, dorsiflexion of the ankle, and this may obscure the toe movement. Hence the importance of a gentle stimulus, graduated so as just to elicit the toe phenomenon and no more.

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[1] Rolleston, *Brain*, 1906, p. 99.

[2] *Neurologisches Centralblatt*, 1905, p. 593.

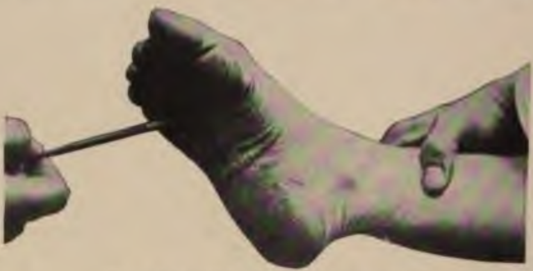

FIG. 144.—Normal plantar reflex.

This normal plantar reflex, flexor in type, only occurs when the reflex arc is intact, and when, in addition, the lower reflex arc is in connection with the cerebral motor cortex by an uninjured pyramidal tract. In this sense it is a "cortical" reflex.

If the pyramidal tract, conveying motor impulses downwards from the cortical "leg-centre," be interrupted in any part of its course by injury or disease, or if it be non-developed, as in little infants who have not learned to walk, the type of plantar reflex is different and is known as *Babinski's phenomenon*, or the *extensor plantar reflex*. In this, stimulation of the sole produces extension of the hallux instead of flexion (see Figs. 144 and 145). Moreover, this extensor movement of the great toe is slower than was the normal, brisk flexion. An extensor plantar reflex is practically always pathological (except in infants too young to walk), and if constantly present it indicates an organic lesion, and one which implicates the pyramidal tract.[1] It is therefore of the utmost value in the diagnosis between hysteria and organic disease.

Somewhat resembling the Babinski reflex, but apparently distinct from it, is Gordon's[2] *paradoxical flexor reflex*, which consists in an extension movement of the great toe or of all the toes when we press deeply, through the calf muscles, on to the deep flexor muscles beneath. To elicit this reflex, the physician should place himself on the outer side of the patient's leg, the muscles of which must be completely relaxed. This is attained either by directing the patient to lie on his back or making him sit with his feet on a stool. The "paradoxical flexor reflex" is never present in health: it occurs only in organic lesions of the pyramidal tract. It is sometimes associated with the Babinski reflex, but may occur in some cases where the Babinski sign is absent.

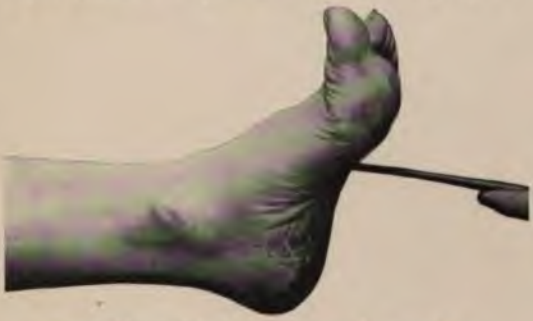

FIG. 145.—Babinski's extensor plantar reflex.

Oppenheim's reflex is an extension movement of the hallux elicited in a slightly different way, viz., by firm stroking with some hard object (such as the handle of a percussion-hammer) just behind the postero-internal border of the tibia, from above downwards. Its significance is the same as that of the paradoxical flexor reflex.

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[1] There is one exception to this rule. This is in the case of a lower motor neurone lesion affecting the flexor muscles of the toes and sparing the extensors. In such a case the only possible movement would be extension. But the other signs of lower neurone lesion, especially the electrical reactions, will prevent error.

[2] *American Medicine*, 1904, p. 971.

The *cremasteric reflex* is very easy to elicit in young children, by stroking the inner side of the thigh. But in old men it is sluggish or apparently absent. Even in them, however, it can usually be elicited by sudden firm pressure backwards against the sartorius muscle in the region of Hunter's canal; this often succeeds when stroking the skin in the usual fashion is of no avail. The cremasteric reflex is frequently exaggerated on the affected side in cases of sciatic neuralgia.

The *bulbo-cavernosus reflex* is of great diagnostic value in determining whether the lowest segments of the spinal cord are intact in a lesion about the level of the third sacral segment—where the ordinary reflexes of the lower limbs give us no help. To obtain the bulbo-cavernosus reflex, we place one finger behind the patient's scrotum, pressing upwards against the bulbous part of the urethra. With the other hand we pinch, or prick with a pin, the dorsum of the glans penis; the bulbous urethra is at once felt to give a brisk twitch. Loss of this reflex indicates a lesion somewhere in its reflex arc, either in the anterior cornua of the third and fourth sacral segments, or in the corresponding motor or sensory roots of the cauda equina. Loss of the bulbo-cavernosus reflex is a fairly common sign in tabes, being due in that case to a lesion of the afferent fibres of the reflex arc.

The *superficial anal reflex* can be easily obtained by pricking the skin of the perineum with a long "bonnet-pin," and watching whether the external sphincter contracts. This reflex is sometimes lost in anæsthesia of the perineum, or in lesions of the fifth sacral or of the coccygeal segment, or of their corresponding motor roots.

Organic hemiplegia usually produces, at first at least, loss of the unilateral superficial reflexes all down the paralysed side, with one exception—viz. the plantar reflex, which persists, but from the first is changed into the extensor type. In hysterical hemiplegia, on the other hand even though cutaneous hemianæsthesia be present, the unilateral skin reflexes on the paralysed side are usually preserved, with the exception of the plantar reflex which is often lost. An extensor plantar reflex never occurs in pure hysteria. In hysteria there may be absence not only of any toe movement on stimulation of the sole, but there may also be loss of the reflex contraction of the tensor fasciæ femoris. This "combined" absence of reflex movement both of the toes and of the fascia lata of the thigh is always strongly suggestive of hysteria.

Deep or Tendon Reflexes.—The following table gives a list of those deep reflexes which we commonly study when investigating cases of nervous disease :—

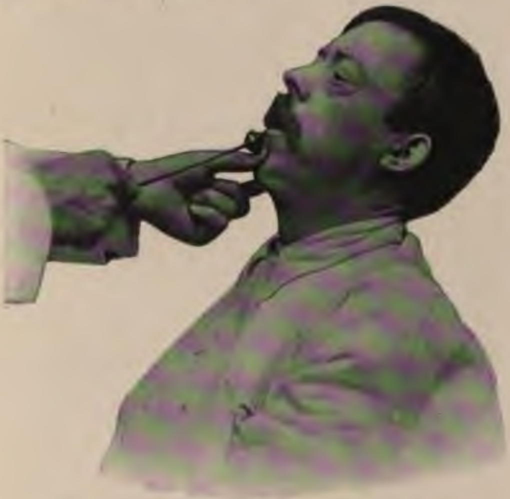

FIG. 146.—Method of eliciting jaw-jerk.

	METHOD OF ELICITING.	RESULT.	SEGMENTAL LEVEL
Jaw	Tapping lower jaw, supported in half-open position.	Jaw closes.	Pons.
Biceps	Tapping biceps tendon.	Biceps contracts.	C5 and C6.
Supinator longus	Tapping styloid process of radius.	Supinator longus contracts.	C5 and C6.
Scapulo-humeral	Tapping vertebral border of scapula, near lower angle.	Teres minor, Infraspinatus, &c., contract.	C5 and C6.
Wrist	Tapping upper part of radius.	Wrist and fingers extend.	C6 to C8.
Triceps	Tapping triceps tendon.	Triceps contracts.	C7 to D1.
Carpo-metacarpal	Tapping dorsum of wrist.	Fingers flex.	C8 and D1.
Knee	Tapping patellar tendon.	Vastus internus, &c., contract.	L3 and L4.
Ankle	Tapping tendo Achillis.	Calf muscles contract.	S1 and S2.

The *knee-jerk* may be taken as a type of these tendon reflexes. It has been a matter of dispute whether it is really a true reflex or not. Strictly speaking, the jerk occurs too soon after the tap, for a nerve impulse to have time to travel up to the reflex centre in the cord and down again to the muscles. But so long as the reflex arc is intact, there is a constant "reflex tonus" in the vastus internus muscle which, when the tendon is struck, permits the jerk to occur. If this reflex tonus be lost from interruption of the reflex arc at any point, the jerk can no longer be obtained. Therefore, for practical purposes, the knee-jerk, though not a true reflex action itself, is an accurate index of the integrity of the reflex arc.

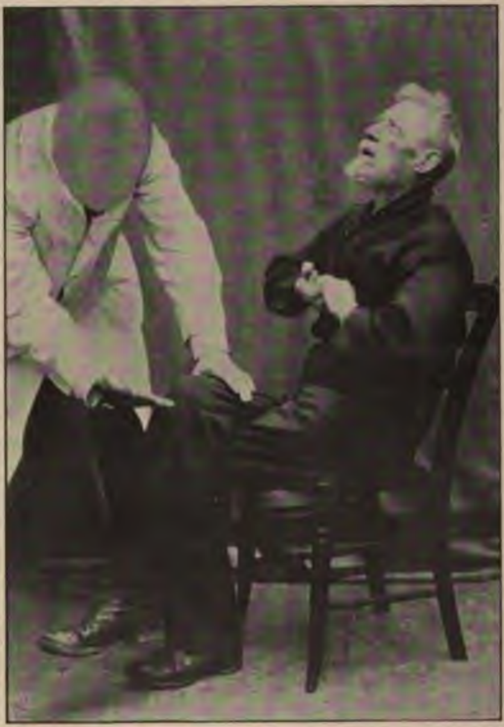

FIG. 147.—Knee-jerk. Reinforcement by Jendrassik's method.

To obtain the knee-jerk, we feel for the patellar tendon and strike it either with the edge of the hand or with some other fairly heavy object, such as a rubber percussion-hammer or a heavy paper-knife. The result is a brisk contraction of the quadriceps. If we grasp the vastus internus with our other hand we can feel the jerk in cases where it is too feeble to move the knee-joint. The knee must be somewhat bent to put the quadriceps slightly on the stretch, and the muscles must be absolutely relaxed. This latter point is of importance; for sometimes it happens that a patient may have his muscles in a state of excessive spasm, so that we may fail to elicit the knee-jerk, and yet, if we succeed in relaxing the muscles by passive movements, the knee-jerk is not only present but found to be increased. The knee-jerk in such a case is not absent, but only "concealed" by the spasticity of the muscles.

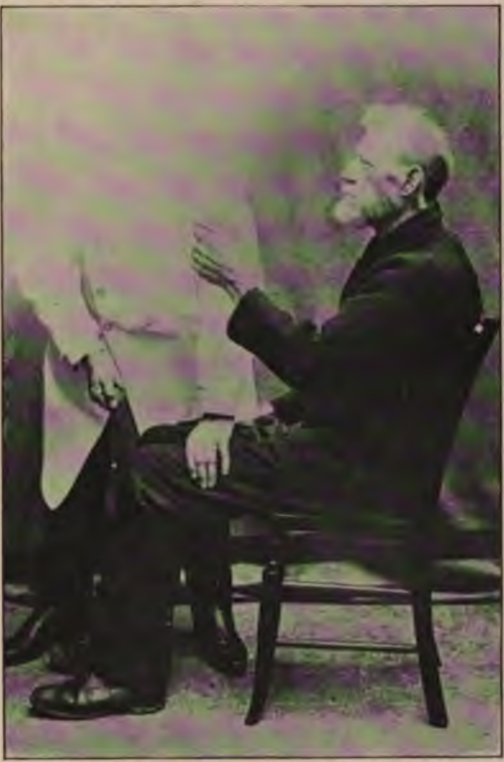

FIG. 148.—Knee-jerk. Reinforcement by Laufenauer's method.

In testing the knee-jerk the patient may be sitting on a table with his legs dangling in the air, or better, on a chair with the soles of the feet flat on the ground and the knees gently semi-flexed, or one knee may be crossed over the other. A feeble jerk may be "reinforced" either by Jendrassik's method (Fig. 147), in which the patient hooks both hands together, pulling them one against the other, and looks up towards the ceiling, thereby diverting his attention and relaxing the leg muscles. An even better method of reinforcement is Laufenauer's, in which we grasp the patient's quadriceps whilst the patient sits with his soles flat on the ground. The patient then grasps our upper arm with one hand, and suddenly squeezes when told to do so; meanwhile, down comes the percussion-hammer (Fig. 148). The advantage of this method is that we can feel for ourselves whether the patient

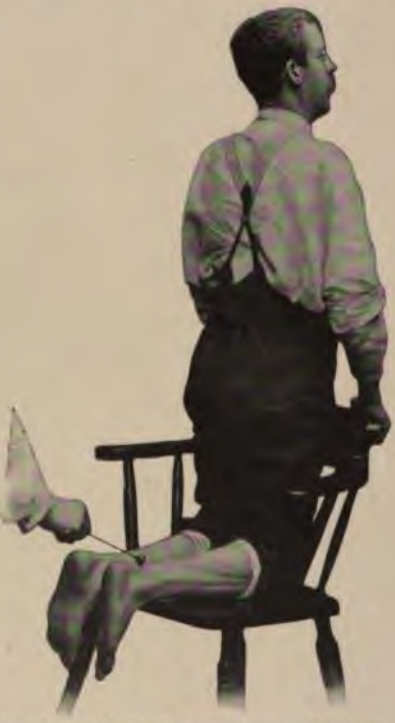

FIG. 149.—Method of eliciting ankle-jerk.

really is directing his attention to the act of reinforcement. Moreover, this method can be employed to reinforce feeble reflexes of the upper limbs, whereas Jendrassik's method is only available for reflexes of the lower limbs.

Reinforcement will make a feeble jerk more evident; but it has no effect if the jerk be absent.

The *ankle-jerk* has a diagnostic significance equal to that of the knee-jerk. To test it, we make the patient kneel on a chair with his feet projecting over the edge, and then tap the tendo Achillis (Fig. 149); a brisk extension movement of the ankle is the result. In tabes the ankle-jerk is often lost before the knee-jerk.

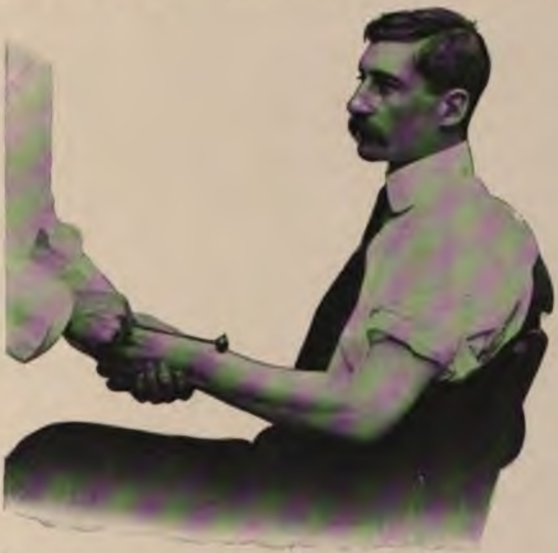

FIG. 150.—Supinator-jerk.

In the upper extremity we have various deep reflexes at our disposal. Of these, the *supinator-jerk* is ordinarily the most accessible. In testing it, we support the patient's hand in a semi-supinated posture, with the elbow loosely bent to a right angle. We tap with our hammer close above the styloid process of the radius (Fig. 150). The supinator longus at once contracts and produces a flexion movement of the elbow. To elicit the *triceps-*

jerk, the best way is not, as stated in some text-books, to allow the elbow to hang loosely over the back of a chair, but rather to support the upper arm horizontally, with the elbow loosely flexed at a right angle. Then, feeling for the triceps tendon, we tap it with our hammer, and the muscle at once contracts.

Loss of Deep Reflexes.—Sometimes these reflexes cannot be elicited, even on reinforcement. This is always pathological, and generally indicates a lesion of the reflex arc. If this lesion be in the afferent limb of the arc, there may also be anæsthesia of the corresponding nerve- or root-area. If it be in the anterior cornu, or in the efferent motor path, there will be muscular paralysis, with atrophy of the particular muscle.

The following is a list of some of the chief conditions in which there is loss of the deep reflexes :—

Disease.	*Site of Lesion.*
Neuritis (Alcohol, Diabetes, Diphtheria, Lead, Arsenic, Tubercle, Cachexia, &c.)	Peripheral nerves, sensory or motor.
Peripheral Nerve Palsies	
Tabes Dorsalis	Posterior columns of cord.
General Paralysis of Insane (tabetic type — Tabo-paralysis	
Friedreich's Ataxia	
Subacute Combined Degeneration of Posterior and Lateral Columns—later stage of	
Any focal lesion in Grey Matter of Cord	Reflex centre in cord.
Infantile Paralysis (Acute Anterior Poliomyelitis)	Anterior cornua of cord.
Progressive Muscular Atrophy (Chronic Anterior Poliomyelitis)	
Amyotrophic Lateral Sclerosis	
Syringomyelia	
Thrombosis of Anterior Spinal Artery	
Landry's Paralysis	Anterior cornua and peripheral motor nerves.
Myopathies (Pseudohypertrophic and Atrophic types)	Muscle itself.
Increased Intra-Cranial Pressure (especially Hydrocephalus and Tumours of Posterior Fossa).	
Pneumonia.	
Family Periodic Paralysis (during attacks).	
Immediately after attack of Major Epilepsy.	
Complete transverse lesion of Cord.	

From this list we see that, in addition to permanent organic lesions within the reflex arc itself, there are other conditions where the deep reflexes are abolished. Thus these reflexes are lost in the final stages of increased intra-cranial pressure, especially in hydrocephalus and in tumours of the posterior fossa. This may perhaps be due to a curious degeneration of the posterior spinal roots, which sometimes occurs in such conditions. The knee-jerks are often lost during pneumonia. In the attacks of the rare disease known as family periodic paralysis (see p. 214), the deep reflexes in the affected limbs are temporarily abolished, owing to temporary paralysis of the muscle-fibres. The deep jerks are also abolished for a few minutes just after an epileptic fit, during the stage of coma and flaccidity. We should also remember that in *complete* trans-section of the spinal cord, all the deep reflexes below the level of the lesion are lost. If, however, the lesion be not a complete one, and there still survives some nervous tissue connecting the upper and lower portions, then the deep reflexes are exaggerated. In both instances, an extensor type of plantar reflex is present.

Exaggeration of Deep Reflexes.—Sometimes the deep reflexes are exaggerated, so that the slightest tap on the tendon produces an unusually brisk contraction. This may be the result of various poisons—*e.g.* tetanus, or strychnine-poisoning—rendering the reflex centres unduly explosive; or it may occur in simple neurasthenia. But organic lesions of the pyramidal tract are by far the commonest causes of permanent exaggeration of the deep reflexes. In cases of organic disease, we look also for the presence of *clonus*. A clonus is a rhythmic series of muscular contractions, produced by sudden passive stretching of the tendon, the clonus continuing so long as the tension of the tendon is maintained.

Ankle-clonus is the commonest clinical variety of clonus. To elicit it, the knee is passively flexed (the angle of flexion varying in different cases), and the ankle is suddenly dorsiflexed by upward pressure on the sole of the foot (Fig. 151). Ankle-clonus is due, as Weir-Mitchell has pointed out, to contraction, not of the gastrocnemius but of the soleus muscle, since in that position of the knee which is best for eliciting ankle-clonus, the gastrocnemius is relaxed.

A *spurious ankle-clonus* is sometimes obtained in cases of hysteria. This can usually be differentiated from genuine clonus by the feeling of voluntary contraction in the muscles, especially at the commencement of the clonus. Spurious clonus is generally poorly sustained and often irregular in rhythm. And it is never associated with an extensor plantar reflex.

Knee-clonus or rectus-clonus is best obtained by sudden downward traction on the patella, the knee being passively extended.

In chronic organic affections of the pyramidal tract, practically every one of the deep jerks may become exaggerated into clonus. Thus in advanced disseminated sclerosis we may find in the same patient jaw-clonus, elbow-clonus, wrist-clonus, finger-clonus, knee-clonus, ankle-clonus, toe-clonus, &c.

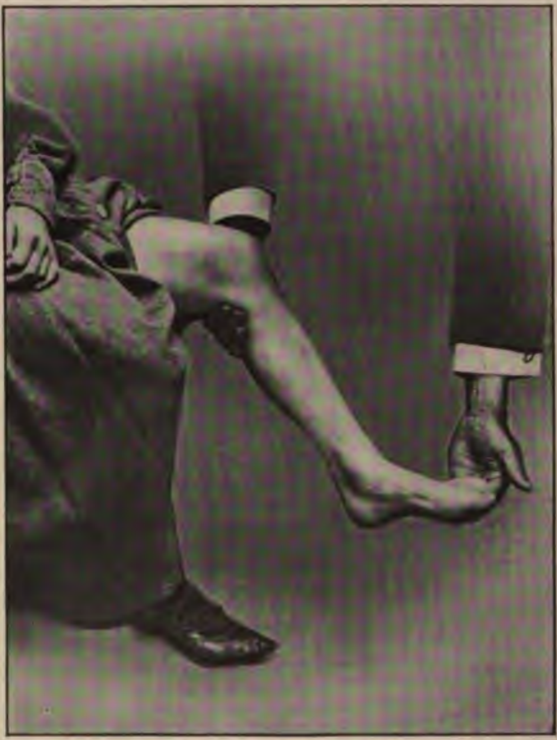

FIG. 151.—Method of eliciting ankle-clonus.

The presence of true clonus indicates that the reflex arc is hyper-excitable, owing to withdrawal of the regulating or restraining influence normally exerted through the pyramidal tract. Exaggeration of the deep reflexes is therefore one of the cardinal signs of a chronic lesion of the upper or cortico-spinal motor neurone.

It is unnecessary to give a list of the various organic diseases in which the deep reflexes are exaggerated. Suffice it to say that any chronic lesion of the pyramidal tract will produce exaggeration of the deep reflexes below the level of the lesion. Thus in paraplegia due to myelitis, to lateral sclerosis, or to some other affection of the lateral columns, or in hemiplegia from organic brain disease, there is exaggeration of the deep reflexes in the paralysed limbs, owing to injury or disease of the pyramidal fibres.

There is an important exception to this rule, to which we have already referred, namely in *total* trans-section of the spinal cord, as in some cases of fractured spine. Such total lesion produces a permanent flaccid paralysis of the lower limbs, with loss of the deep reflexes. But if the lesion be incomplete, we have the usual spastic type of paraplegia, with increased deep reflexes. In both instances, whether the lesion be total or partial, we have an extensor type of plantar reflex.

We should also mention that in the early stages of *peripheral neuritis* the deep reflexes may be exaggerated. But this soon passes off and is succeeded by their diminution and loss. Again, in *subacute combined degeneration* of the lateral and posterior columns of the cord, there is an early stage of spasticity with increased deep reflexes, and a terminal stage of flaccid paralysis with loss of reflexes.

Sometimes in spastic paraplegia the muscular rigidity of the paralysed limbs is so excessive that it may be almost impossible to obtain the knee-jerks and other deep reflexes, which we should ordinarily in these cases expect to find exaggerated. This is because the muscles are already in a state of tonic spasm. But if we succeed in temporarily relaxing them, by passive changes of posture, we can sometimes obtain the increased jerks and even the clonus. And in most cases, the spasticity and the presence of an extensor plantar reflex will prevent errors in diagnosis.

Pupillary Light Reflex.—This occupies a special category of its own. The reflex is elicited by exposing the pupil to light, after previous shading. Normally under such conditions the pupil contracts briskly, both when stimulated directly and when the pupil of the opposite eye is exposed to light (consensual reflex).

In some respects the pupil reflex to light, although occurring in a non-striated muscle, is analogous to a deep reflex, and it is useful to remember that in tabes this reflex disappears, as do the ordinary tendon-reflexes. But the subject of the various pupil-reflexes and their connections with the third nerve, with the ciliary ganglion, and with the cervical sympathetic are discussed elsewhere (see "Cranial Nerves," p. 105, and "Cervical Sympathetic," p. 287).

Organic Motor Reflexes.—These are concerned with the sympathetic nervous system and with the contraction of non-striated, involuntary muscles. Non-striated or smooth muscles contract slowly, unlike the brisk twitch of a reflex in a striated muscle. The following is a list of organic reflexes which are of diagnostic interest. In some of these, such as the cilio-spinal or the scrotal, the reflex movement is executed entirely by non-striated muscle. In others, such as the vesical, uterine, or rectal, the non-striated muscle is reinforced by voluntary striated muscles.

	METHOD OF ELICITING.	RESULT.
Cilio-spinal .	Pinching or scratching skin of neck.	Pupil dilates.
Scrotal . . .	Repeated stroking of perineum or application of cold.	Dartos contracts.
Vesical . . .	Distension or irritation of bladder or posterior urethra.	Bladder-wall contracts.
Rectal . . .	Distension or irritation of upper part of rectum.	Rectum contracts.
Genital . . .	From cerebrum or periphery.	Erection of corpora cavernosa.
Uterine . . .	Distension or stimulation of uterus.	Uterus contracts.
Internal anal .	Distension of anus by finger.	Internal sphincter ani contracts.

In all of them, with the exception of the cilio-spinal, the reflex movement can be accomplished, more or less perfectly, independently of the central nervous system.

Formerly the reflex centres for the bladder, uterus, and rectum, were regarded as situated within the cord; and even yet in some of the text-books they are so located. But within recent years clinical and pathological evidence has been brought forward, notably by L. R. Müller,[1] showing that the lowest reflex centres for the contraction of the bladder, and of the neighbouring hollow viscera possessing non-striated muscular walls, are situated extraspinally, in the hypogastric and hæmorrhoidal plexuses of the sympathetic.

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[1] *Deutsche Zeitschrift für Nervenheilkunde*, 1901, Band 21, s. 86.

The sympathetic vesical centre can be stimulated from the cerebro-spinal system. Micturition in the adult is a voluntary act, but only to this extent that it can be voluntarily initiated. This is accomplished by contracting the abdominal walls, producing a rise in the intra-vesical tension, which starts the reflex; meanwhile the striated constrictor urethræ is voluntarily relaxed. But the non-striated bladder-wall itself, the so-called detrusor urinæ, is not under control of the will. Once started, the bladder empties itself spontaneously, and we can stop the act only by forcibly innervating the constrictor urethræ, usually a matter of considerable effort. But reflex micturition is often excited by irritation of the urethra, especially of its vesical end. Thus if a few drops of urine trickle into the prostatic urethra, an imperious reflex act of micturition results, which is difficult to prevent. Similar phenomena are produced by the irritation of a posterior urethritis.

The fibres from the brain and spinal cord to the sympathetic vesical centre reach it through the lowest spinal roots, so that lesions of the spinal cord or cauda equina constantly cause bladder trouble. This generally takes the form of initial *retention* of urine, followed after several days by *reflex incontinence* ("incontinenza a getto" of Italian writers [1]), in which the bladder contracts intermittently and expels the urine at intervals. Such reflex incontinence is generally associated with incomplete emptying, so that a certain amount of "residual urine" remains in the bladder. In cases of coma or of the deep insensibility of fevers such as typhoid, we observe an initial retention of urine, followed by distension, paralysis of the bladder-wall and overflow dribbling ("incontinenza per regurgito").

The sensory nerves from the bladder pass through the sympathetic by the rami communicantes, and along the posterior roots into the spinal cord. They serve to inform us of the distension of the bladder. There are also higher micturition centres, some in the spinal cord, others higher still in the brain, in the corpus striatum and optic thalamus; others, highest of all, in the motor cortex, between the arm and leg centres, controlling the lower centres. When these higher centres are hyper-excitable, whether from disease or from emotion, we may have *precipitancy* of micturition, or even enuresis, a condition in which the brain and spinal cord on the slightest provocation send impulses which relax the compressor urethræ. A similar condition exists in little infants who have not learned to control their subcortical micturition centres.

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[1] Rebizzi, *Rivista di Patologia Nervosa e Mentale*, 1905, p. 80

True dribbling of urine ("incontinenza vera"), as distinguished from intermittent contraction of the bladder, occurs most typically in cases of tabes and is due to anæsthesia of the bladder, which being now insensitive to distension, is no longer stimulated to contract by the normal accumulation of urine. The tabetic patient with an anæsthetic distended bladder expels his urine, not by contraction of the bladder but by pressure with his abdominal walls. This can readily be verified if we have to pass a catheter for the purpose of emptying a tabetic patient's bladder. True dribbling also occurs in an over-distended bladder which, from obstruction in the prostate or urethra, has become flaccid, paralysed and atonic.

The *internal anal reflex* is tested by inserting a finger within the anus. Normally the finger is tightly grasped by the non-striated internal sphincter. This reflex is independent of the superficial anal reflex of spinal origin, to which we have already referred. When the internal anal reflex is lost, the anus no longer grasps the finger but remains open for several seconds, "yawning," after the finger has been withdrawn. Such loss is most commonly due to anæsthesia of the anus, as in tabetic or other lesions of the cauda equina. The result is incontinence of fæces. If there be a lesion within the spinal cord, above the spinal centre in the conus medullaris, there is intermittent rectal incontinence. But if the lesion be in the afferent nerves from the rectum, the internal sphincter remains relaxed, and the fæces, if fluid, dribble away continuously whenever they enter the rectum, the patient being unconscious of the fact.

To test the *scrotal reflex*, which is an excellent example of a purely sympathetic motor phenomenon, the patient stands bending forwards with his legs wide spread and scrotum hanging free. The skin of the perineum is now stroked with some hard object five or six times in succession. After a few seconds an extremely slow, worm-like contraction appears in the non-striated dartos muscle, beginning near the perineal part of the scrotum and spreading forwards. This reflex can also be elicited by the application of cold to the perineum or scrotum.

Another good example of a non-striated muscle reflex is Mackenzie's *pilo-motor reflex*.[1] This is elicited in many people by rubbing the skin firmly just beneath the nipple with a piece of flannel or giving a couple of brisk strokes to the chest with a rough towel. As a result, the pilo-motor muscles of certain hair-follicles contract, producing a local "goose-skin" or cutis anserina. This area of goose-skin may spread up to the second rib, and down the inner border of the upper limb as far as the little finger and adjacent side of the ring finger, in the areas of the posterior roots of the eighth cervical and two or three upper dorsal segments of the cord (see Fig. 16, p. 28). When this reflex appears, it is often accompanied by a peculiar local sensation of cold—which Mackenzie calls the "autonomic sensation"—in the goose-skin area, and by dilatation of the pupil on the same side, the so-called *autonomic pupil-reflex*. These various phenomena are produced through nerve-paths which traverse both the spinal cord and the cervical sympathetic.

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[1] *Brit. Med. Journal*, June 23, 1906, p. 1449; also *Brain*, 1893, p. 515.

LECTURE XX

AFFECTIONS OF THE SYMPATHETIC. ANGIO-NEUROSES

To most of us the mention of the Cervical Sympathetic recalls memories of our early days as students of physiology, and more particularly the memory of a classic experiment of Claude Bernard upon the rabbit. But it is perhaps not sufficiently realised that the cervical sympathetic is also of considerable clinical importance.

First of all, let us recall a few anatomical points. The sympathetic nervous system forms two gangliated cords, hanging like two strings of beads, one on each side, close in front of the vertebral column, and extending from the base of the skull to the front of the coccyx. Above, these chains are connected with plexuses which enter the cranial cavity; below, they converge and end in a loop on the coccyx. Each chain is made up of multipolar nerve-cells and nerve-fibres, all of them involuntary fibres. In addition to its own longitudinal fibres, the sympathetic chain receives connecting-fibres from the central nervous system. These are called the *rami communicantes*—some of them white, some of them grey. They join the sympathetic to the anterior primary divisions of the spinal nerves. The white *rami*, consisting of medullated fibres, pass from the spinal cord to the sympathetic ganglia; they are all efferent in function, and leave the cord through the anterior nerve-roots. The grey *rami*, consisting of non-medullated fibres, originate within the sympathetic chain itself, and, like the others, join the spinal nerves. Some of them are afferent, going into the spinal cord, reaching it through the posterior nerve-roots; others turn off with the peripheral nerves and supply to the skin involuntary efferent fibres, whose functions are vaso-motor, vaso-inhibitory, pilo-motor, secretory, &c. In addition, the sympathetic cords give branches, either directly or through the great prevertebral ganglia (the cardiac, solar, and hypogastric plexuses), to the various glands and viscera of the body, to the heart and blood-vessels, to the genital organs, and to the non-striped muscles of the body generally.

Lastly, the cervical part of the sympathetic chain has very special "oculo-pupillary" fibres, which are clinically of considerable importance. These supply the dilator pupillæ, the non-striated part of the levator palpebræ superioris and the orbital muscle of Müller—a small bundle of non-striated muscle which lies behind the globe of the eye and bridges across the spheno-maxillary fossa at the back of the orbit. The cervical sympathetic also supplies secretory fibres to the submaxillary gland, and, like the sympathetic elsewhere, it supplies fibres to the cutaneous

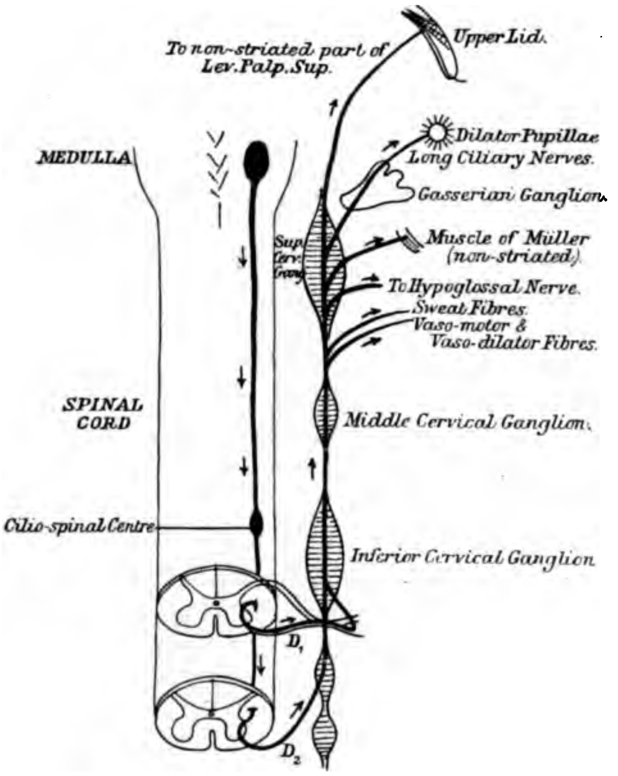


FIG. 152.—Diagram of course of oculo-pupillary fibres of cervical sympathetic.

blood-vessels, also (through the hypoglossal nerve) to the vessels of the tongue, and, lastly, fibres to the sweat-glands of the head and neck.

The pupil-dilating fibres have a peculiar course, which it is important to remember (see Fig. 152). Arising from the pupil-dilating centre in the medulla, they descend in the lateral column of the spinal cord to the cilio-spinal centre in the lower cervical region. They emerge from the cord through the anterior roots of the first and second dorsal segments, and enter the inferior cervical ganglion of the cervical sympathetic by white *rami communicantes*. They then ascend in the cervical sympathetic to the Gasserian ganglion and pass thence to the orbit (along the ophthalmic division of the fifth cranial nerve), and *viâ* the long ciliary nerves to the pupil. They do not traverse the ciliary ganglion (see Fig. 39, p. 104).

It is evident that ocular and other symptoms may be produced not only by lesions of the ascending fibres of the cervical sympathetic, but also by lesions within the cord, affecting the fibres in their downward course from the medulla (this being remarkably frequent in syringomyelia and in unilateral lesions of the medulla), or by lesions of the first two dorsal nerves or of their anterior roots, or even by lesions of the ophthalmic division of the fifth cranial nerve, though this last is rare.

The signs of **paralysis of the cervical sympathetic** are very characteristic. Fig. 153 is that of a woman who was sent to hospital with the history that six years previously she had some tuberculous glands removed from the right side of her neck. When she recovered from the anæsthetic, she learned that the jugular vein had been injured during the operation. But other structures had also been injured, and amongst them was the cervical sympathetic (which lies behind the carotid sheath), because ever afterwards, she noticed that the right eyelid drooped a little, that the right side of the face flushed less than the left, and that when she chewed, a small patch of excessive perspiration appeared below the right eye. We observe from the photograph that the right pupil is smaller than the left, from paralysis of the dilator pupillæ.

Moreover, the affected pupil does not dilate when shaded, but it contracts briskly to light and on convergence, since the third cranial nerve, which through the ciliary ganglion innervates the sphincter pupillæ, is unaffected. Further, we notice that the upper lid droops, making the palpebral fissure narrower than on the healthy side. This is due to paralysis of the non-striated part of the levator palpebræ, which is inserted into the upper edge of the tarsal cartilage. The voluntary, striated fibres of the levator, inserted into the skin of the upper lid and supplied by the oculomotor nerve, are unaffected, and the patient is therefore able to elevate the lid voluntarily to its full extent. This, therefore, is not a true ptosis, but a "pseudo-ptosis."

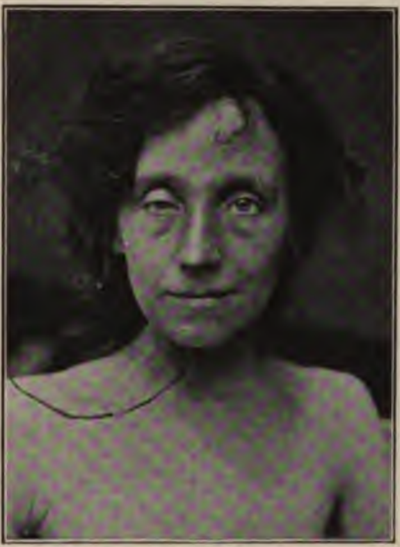


FIG. 153.—Paralysis of cervical sympathetic on the right side. The black line encloses an area of anæsthesia, due to division of cutaneous nerves.

We also notice that the right eye has sunk into the orbit, owing to paralysis of the non-striated muscle of Müller, which normally keeps the globe pressed forwards. This "enophthalmos" narrows the palpebral fissure still more. The difference in antero-posterior projection between the two eyes is seen best if we make the patient lie down, and then we stand behind and look down at the forehead and eyeballs from above.

On palpating the two globes, we find that the intra-ocular tension is diminished on the affected side.

Such are the "oculo-pupillary" symptoms of cervical sympathetic paralysis. There are, however, one or two additional points. When the cervical sympathetic is paralysed, it no longer responds to stimulation. There are two clinical ways of stimulating it. One is by pinching or pricking the side of the neck, when we produce a dilatation of the pupil on the same side; this "*cilio-spinal reflex*" is abolished in cervical sympathetic palsy. Another method of stimulating the cervical sympathetic is to drop into the conjunctiva a few minims of a solution of cocaine. The result is that the pupil dilates, the upper lid retracts, and the eyeball is pushed slightly forwards. All these phenomena were absent in the patient just referred to. She also told us that when her face flushes, it does so only on the affected side. Moreover, her face on the affected side no longer sweats except in one little patch below the

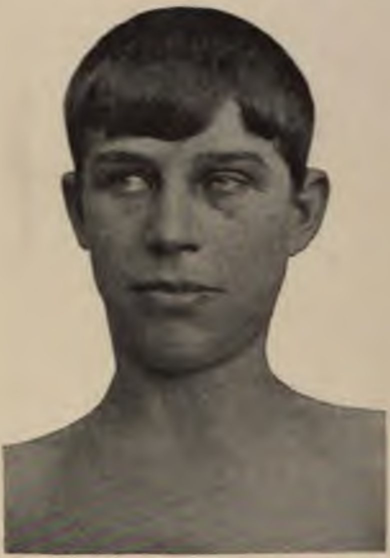


FIG. 154.—Paralysis of the left cervical sympathetic from a tumour at the root of the neck.

orbit, where it sometimes sweats spontaneously when she chews. To verify this point we made her sweat profusely by means of pilocarpin, and found that the right side of the face remained dry, except in a small area below the inner canthus of the eye. This survival of a little oasis of sweating on the dry side might perhaps mean that a small twig of the sympathetic had escaped injury, or more probably that sweat fibres to that part of the face are supplied through a branch of the trigeminal nerve.

So much for lesions of the sympathetic chain itself. But the oculo-pupillary and other fibres may also be damaged at some point between the spinal cord and the inferior cervical ganglion. Fig. 154 is that of a boy of seventeen who had felt a tingling sensation down his left arm for several months. A few weeks before he came under observation, he noticed that a swelling had appeared in the lower part of the neck on the same side. At a glance we see that the cervical sympathetic is affected. There is pseudo-ptosis, myosis, and enophthalmos. The left cheek is a little fuller than the right. The cilio-spinal reflex was absent on the left side. On examining the root of the neck, we found that the left clavicle was bulged forwards at its inner end, the supra-clavicular fossa being filled up. Further, there

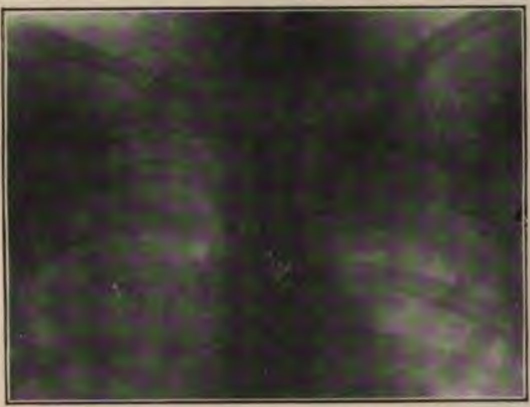


FIG. 155.—X-ray photograph, showing tumour at apex of left lung, in patient represented in Fig. 154.

was percussion-dulness all over the apex of the left lung, with diminution of breath sounds and of vocal resonance. There was also a strip of diminished sensation to touch and pain along the inner side of the left upper limb, reaching to the wrist and corresponding to the cutaneous areas of the first and second dorsal roots. Moreover, there was slight wasting of the hypothenar muscles of the left hand, supplied by the first dorsal anterior root. The skin of the left hand was dry, whilst that of the right was moist, and the patient himself noticed that in hot weather the right side of his face sweated alone. The pulse in the left upper limb was smaller than in the right, on comparing corresponding arteries of both sides. All this pointed to the presence of a solid mass behind the left clavicle, compressing the subclavian artery, affecting the first and second dorsal nerves, including not only their sensory and motor fibres but also the cervical sympathetic fibres. This diagnosis

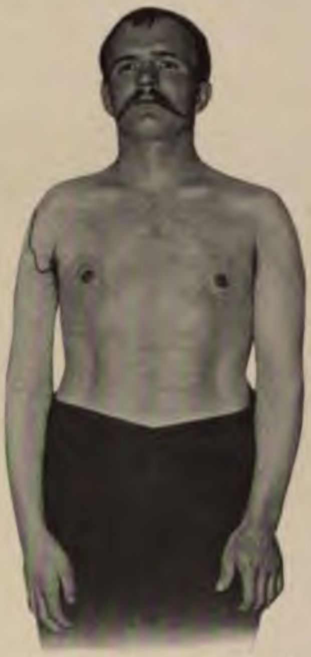


FIG. 156.—Rupture of brachial plexus on right side. Total anæsthesia of right upper limb below black line.

was confirmed by radiography, which showed very clearly (Fig. 155) a tumour at the apex of the left lung. This tumour grew rapidly, compressed the subclavian vein, producing œdema of the left upper limb, and within six months the patient died.

Fig. 156 is that of a seaman on an Atlantic liner who was thrown by a heavy sea against an iron rail, rupturing the whole of the brachial plexus on the right side. He was unconscious for a number of days, and during this period he was trephined over the left Rolandic area on the supposition that the paralysis of the arm was of cerebral origin. In addition to complete motor and sensory paralysis of the upper limb obviously of lower motor neurone type, resulting from the brachial plexus palsy, he showed very beautifully the oculo-pupillary signs of cervical sympathetic paralysis on the right side—enophthalmos, myosis and pseudo-ptosis (see Fig. 157). In this patient, however, the pupil still dilated to cocaine, probably because some pupil-dilating fibres, entering the cervical sympathetic from the second dorsal root, had escaped injury.

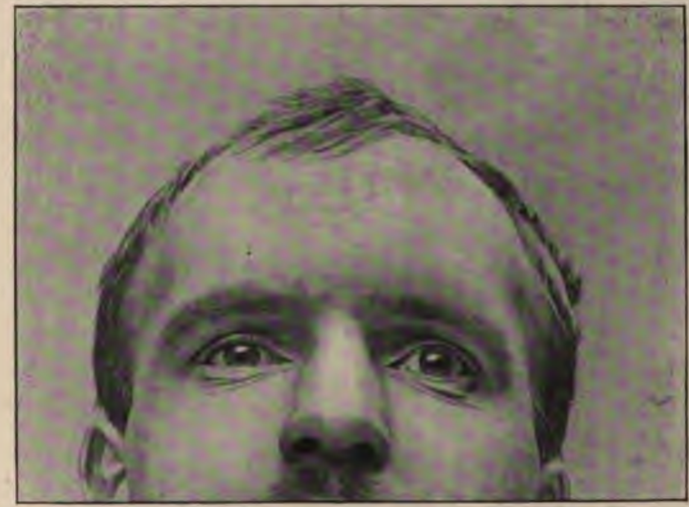


FIG. 157.—Paralysis of right cervical sympathetic from rupture of brachial plexus.

Figs. 158 and 159 are those of a soldier who came under my observation during the South African war. He had been wounded in the neck by a Mauser bullet. At the time of his injury he was lying on his face, firing at the enemy. The bullet entered his neck an inch and a half below the left mastoid process, crossed the middle line in front of the vertebral column, and came out through the seventh right interspace in the posterior axillary line, lodging in his bandolier. He immediately felt a sensation "like an electric shock" all over his body, but especially in the right upper extremity, which became at once totally paralysed. He had hæmoptysis owing to the perforation of the lung, and for a few days some difficulty of swallowing, probably due to injury of the œsophagus. In about three weeks the hæmoptysis gradually ceased, and the right upper limb recovered power, so that when I first saw him, two and a half months after the injury, he was

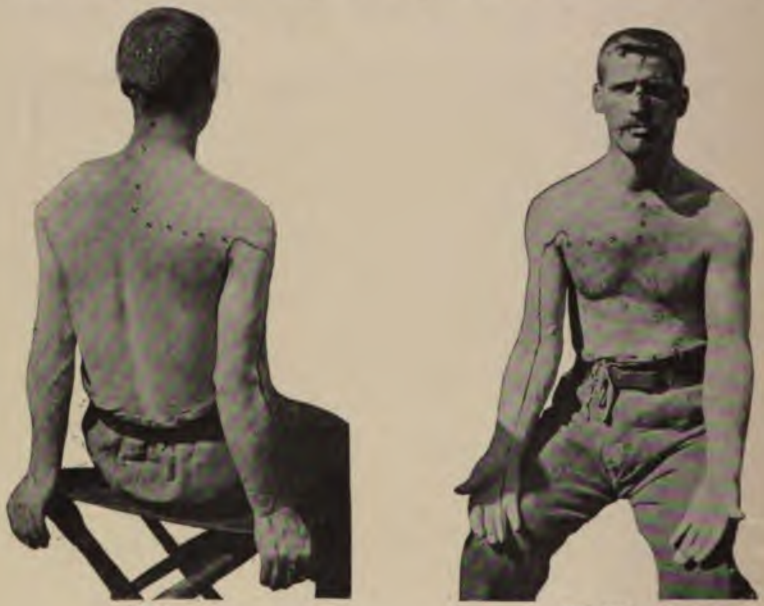


FIG. 158. FIG. 159.

Figs. 158 and 159.—Paralysis of the right cervical sympathetic, from a bullet wound of the lower roots of the brachial plexus. The area of the right upper limb within the black line is anæsthetic. The crosses on the trunk indicate the boundary of an area of anidrosis.

able to move it freely at all joints. Ever after the accident he noticed that he did not sweat on the right side of the face and neck, nor in the right upper limb.

The photographs show that the patient has an area of slight analgesia (bounded by the thick black line) along the inner border of the right upper limb, corresponding to the areas supplied by the eighth cervical, first dorsal, and second dorsal nerve-roots. He also has slight weakness of the small muscles of the thumb, innervated by the first dorsal root. We also observe that the right cervical sympathetic is paralysed, so that he has contraction of the pupil, enophthalmos, and pseudo-ptosis on that side.

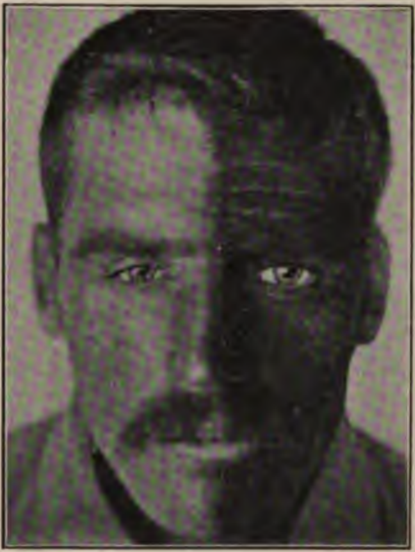


FIG. 160.—Paralysis of the right cervical sympathetic, showing oculo-pupillary phenomena and area of anidrosis.

But there is a point of special interest in this patient's photographs. They demonstrate a point which, so far as I know, had not previously been mapped out in the human subject, viz., the extent of skin supplied with sweat-fibres by the cervical sympathetic. In the tropical heat to which we were exposed, this patient sweated profusely, except in an area on the right side of the head, neck, upper limb, and upper part of the trunk. That area remained dry, and the boundary between sweating and non-sweating skin was sharp and distinct. In order to photograph it, the happy thought occurred to blow powdered charcoal on the skin. This stuck on the sweating side, and blew off on the dry side. We were then able to photograph the non-sweating area, to which the cervical sympathetic should have been distributed. As may be seen from the photographs (Figs. 158 to 160), the boundary of this area, marked by crosses, runs down the middle line of the head and neck, and turns horizontally across the chest at the level of the third rib in front and the spine of the scapula behind, including the whole of the upper limb.

Before leaving the subject of cervical sympathetic palsy, it should be mentioned that excision of the cervical sympathetic has been employed therapeutically in certain diseases. Thus in cases of glaucoma, ophthalmic surgeons have recommended this operation to diminish the intra-ocular tension, and a certain amount of benefit has resulted. Bilateral excision of the cervical sympathetic has also been tried for the relief of epilepsy, in the hope of paralysing the cerebral vasomotor nerves and thereby rendering the brain hyperæmic, especially in cases where fits were ushered in by blanching of the face. But the results recorded have not been sufficiently good to warrant us in recommending this procedure.

Let us now consider briefly the reverse condition, viz., **irritation or stimulation of the cervical sympathetic.** The signs are exactly the converse of those produced by paralysis. We therefore have dilatation of the pupil, exophthalmos or forward projection of the eyeball, widening of the palpebral aperture (Stellwag's sign), and delayed descent of the upper lid when the patient looks downwards (von Graefe's sign). All these phenomena can be produced, to a certain degree, by dropping into a healthy eye a solution of cocaine, which stimulates the cervical sympathetic.

Fig. 161 is that of a man who had lymphadenomatous enlargement of the lymphatic glands. During his stay in hospital, we noticed that quite rapidly, within a day or two, he developed on the right side exophthalmos, widening of the palpebral fissure, and some dilatation of the pupil. There was also in hospital at the same time a soldier who had similar signs in one eye due to an aneurism at the root of the neck, pressing on and irritating the cervical sympathetic. If such pressure continues, the signs of stimulation may pass off and be replaced by those of paralysis. Our lymphadenomatous patient, however, died nine weeks after the appearance of stimulation phenomena, without any change in the direction of paralysis.

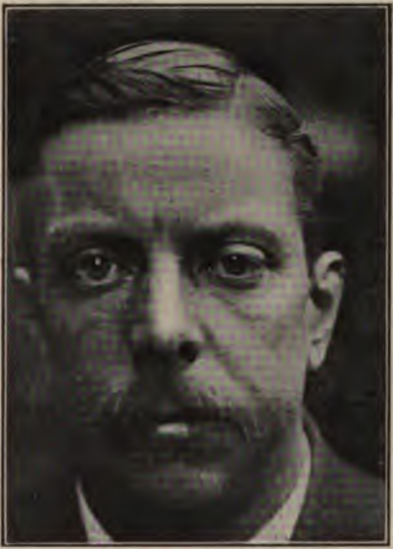


FIG. 161.—Stimulation of the right cervical sympathetic.

Certain cases of apical phthisis are accompanied by inequality of the pupils, probably due to irritation of the cervical sympathetic by changes in the pleura. There is a good deal in favour of the view that many of the signs of exophthalmic goitre are the result of stimulation of the sympathetic, whether by excessive or perverted thyroid secretion. Figs. 162 and 163 are from a case of exophthalmic goitre where the symptoms were preponderatingly right-sided. The tachycardia of this disease is probably due to stimulation of cardio-accelerator fibres, which we know are derived from the sympathetic. The thyroid enlargement may perhaps be explained as a result of vasomotor paralysis of the cervical vessels, including those of the thyroid gland itself.

But though affections of the cervical sympathetic are the most readily recognised, we must not forget that the **thoracic and abdominal portions of the sympathetic** may also be diseased, although the symptoms thereby produced, being mainly visceral, are less easy of diagnosis. Thus some cases of angina pectoris may be the result of irritation of the cardiac plexus, whilst the peculiar reflex vaso-constriction of the pulmonary vessels, with its transient dilatation and irregularity of the right heart met with in certain gastric and hepatic disorders, has been explained by an affection of the thoracic part of the sympathetic. Affections of the abdominal sympathetic or of its great pre-vertebral ganglia

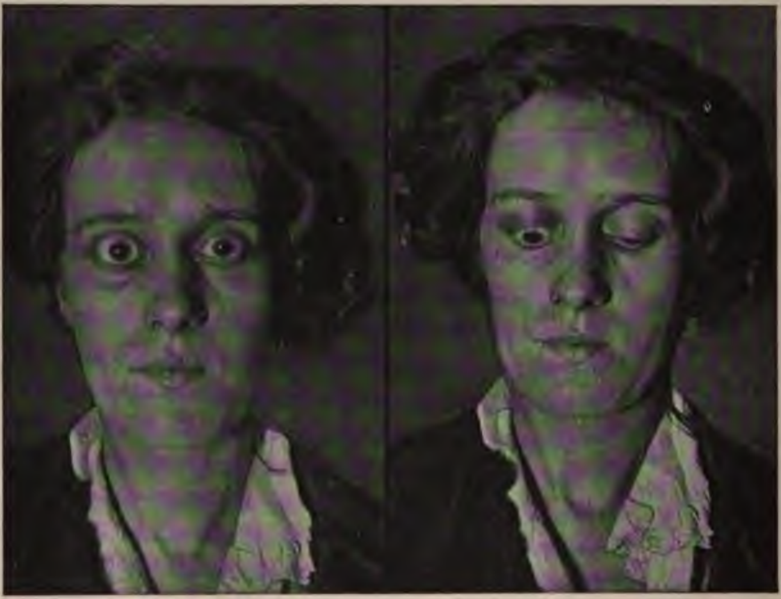


FIG. 162. FIG. 163.

Exophthalmic goitre, the ocular symptoms being more marked on the right side. Fig. 163 shows von Graefe's sign in the right eye on looking downwards.

produce still more striking clinical phenomena. Thus the rare condition known as acute dilatation of the stomach, and many of the symptoms of acute peritonitis, such as the intestinal paralysis, meteorism, small pulse, and general collapse, may be the result of acute paralysis of the solar plexus—the "abdominal brain"; whilst irritation of the solar plexus is exemplified in cases of lead colic, with its pain, constipation, and increased arterial tension. The various visceral "crises" of tabes—gastric, intestinal, renal, &c.—may also be due to irritative changes in the various parts of the abdominal sympathetic. To the same cause also may be attributed the intestinal symptoms of exophthalmic goitre, consisting not in ordinary diarrhœa with loose motions, but rather in an abnormal frequency of defæcation. Muco-membranous colitis, with its characteristic paroxysms, has been ascribed by Mathieu and others to some disorder of the solar plexus, though pathological evidence on the point is as yet scanty. To solar or splanchnic disease may also, perhaps, be attributed such affections as orthostatic albuminuria, diabetes insipidus, and certain forms of glycosuria.

In addition to visceral disorders, disease of the abdominal sympathetic is also associated with certain pigmentary changes in the skin. Of these, the most striking examples are furnished by the cutaneous pigmentation which is occasionally present in exophthalmic goitre and, still more, by Addison's disease, with its characteristic bronzing of the skin, its asthenia and its paroxysmal diarrhœa. Addison's disease is probably the result of two factors—irritation of the abdominal sympathetic and inadequacy of suprarenal function, the proportional rôle played by each of these varying in different cases.

Angio-Neuroses.—These comprise a group of diseases which appear to depend on disorders of the sympathetic fibres which regulate the blood-vessels. The paroxysmal dyspnœa of *asthma*, with its slow and laboured breathing, is doubtless to be referred to a neurosis of the thoracic sympathetic, whether, as some hold, the phenomena be the immediate result of a sudden vascular engorgement of the bronchial mucous membrane, or whether produced, according to another view, by spasm of the non-striated bronchial muscles. In any case, the paroxysmal nature of the affection, its apparent toxic origin in some cases, its association in other cases with nasal or other reflex sources of irritation, its frequent association with the gouty diathesis—all these facts would indicate a functional and not an organic lesion of the sympathetic.

Most angio-neuroses, however, affect the blood-vessels of the more superficial parts of the body, such as those of the skin or of the muscles. Amongst the cutaneous angio-neuroses, perhaps the commonest is the syndrome known as *Raynaud's disease.* The mildest degree of this is *local pallor* ("local syncope" or "dead finger"), where the affected parts, usually the fingers, less frequently the toes, the edges of the ears, or the tip of the nose, suddenly become cold to the touch and of a waxy white colour. Together with this, the patient feels a tingling or other peculiar sensation. Actual blunting of sensation to touch may be present. One or both hands may be affected, sometimes the identical fingers in both hands; the thumb is less often affected than the other digits. The attacks last from a few minutes to several hours and are commonest in winter. They are often brought on by washing the hands in cold water, or by fine digital movements such as sewing or piano-playing. As the attack passes off, the patient feels a sensation of tingling or even of pain. A more severe variety is that of *local asphyxia* or cyanosis, in which the affected digits suddenly become discoloured, varying in tint from a dusky blue or slate colour to an intense purplish-black. Pressure on the discoloured area causes a white mark which persists for several seconds, before the lividity slowly reappears. The pain is usually more intense than in local syncope. As the attack passes off, the affected part often sweats freely. But the most severe variety of Raynaud's disease is *symmetrical gangrene*, which is usually preceded by local asphyxia and sometimes by local syncope. As a rule, the gangrenous process is confined to a small part of the cyanosed area. In its mildest form the necrosis is limited to the epithelium, so that only desquamation results; more often a small blister forms, with blood-stained contents. This bursts, leaving an ulcer which subsequently cicatrises. The nails may be lost and subsequently reproduced. Or the necrosis may extend deep into the tissues, forming a dark slough. Whole phalanges may undergo dry mummification and be cast off, leaving a conical stump. The bones as a rule escape necrosis. Pain is usually severe during the initial cyanosis. The symptoms of Raynaud's disease are due to local spasm of the vessels, the sudden onset and disappearance of the symptoms being incompatible with any other hypothesis. The coexistent sensory symptoms are probably due to imperfect blood-supply. A paroxysm of local syncope or cyanosis can usually be relieved, as Cushing has pointed out, by applying a tight flat rubber tourniquet around the limb above, so as to occlude all the vessels, both arteries and veins, and leaving it on for several minutes. Then, when the tourniquet is taken off, there is a temporary vasomotor paralysis, the whole limb flushes to the finger-tips, and a wave of redness wipes out the local pallor or cyanosis. Raynaud's disease is sometimes associated with paroxysmal hæmoglobinuria, probably due to vasomotor spasm of the renal vessels. In rare cases paroxysmal impairment of vision has been noted, and during such attacks the retinal arteries on ophthalmoscopic examination have been seen to be spasmodically contracted.

In striking contrast with Raynaud's disease is the condition known as *erythromelalgia* (see Plate II.), which usually attacks one or other foot. There is pain of a burning or stabbing character, often of excruciating severity, occurring in paroxysms lasting from a few minutes to several hours. This pain is always aggravated by a dependent posture of the limb, by voluntary movement, or by warmth, whilst it is diminished by a horizontal or elevated posture, by rest, and by cold applications. The earliest attacks consist simply of pain. As the disease progresses, however, redness and swelling of the foot are superadded; more rarely, redness precedes pain. The redness is of a bright tint, often confined, for instance, to the ball of the big toe or to small areas of the sole or edge of the foot. Sometimes it is diffused over the foot. The redness increases in intensity, and if the limb be allowed to hang down, the "vascular storm" continues, with bounding arteries, local rise of temperature, and intense cutaneous hyperæsthesia. As the attack subsides the redness is replaced by cyanosis. In one case which I observed, desquamation of the affected area occurred after each paroxysm.

But there are other cases in which the vasomotor symptoms are intermediate in type between the two extremes of Raynaud's disease and erythromelalgia. These two diseases may coexist, or may succeed each other in the same patient. Erythromelalgia is sometimes one of the earliest signs of organic cord disease, such as disseminated sclerosis, and this would point to its origin from the spinal vasomotor centres, perhaps in the "intermedio-lateral" group of nerve-cells between the anterior and posterior horns,

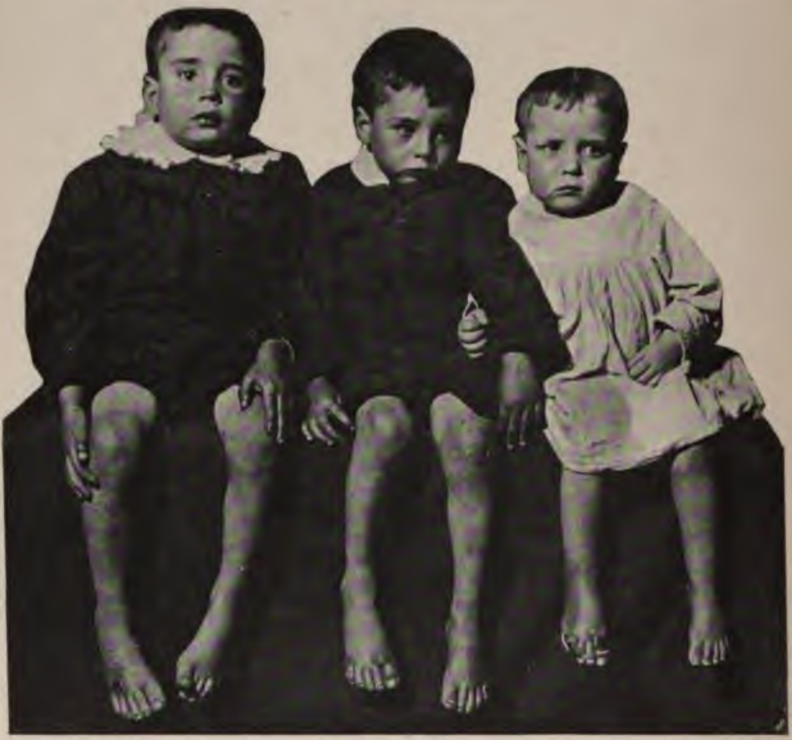


FIG. 164.—Family gangrene in three brothers, affecting the feet.

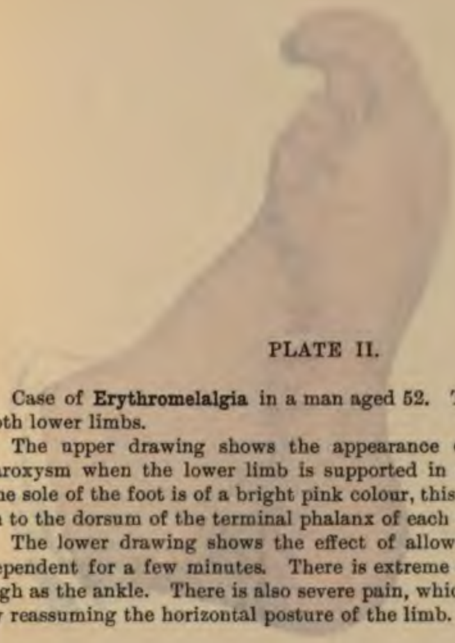


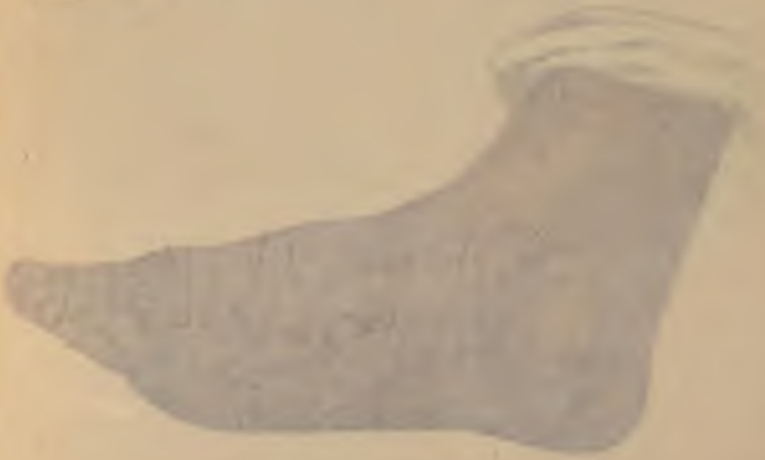
PLATE II.

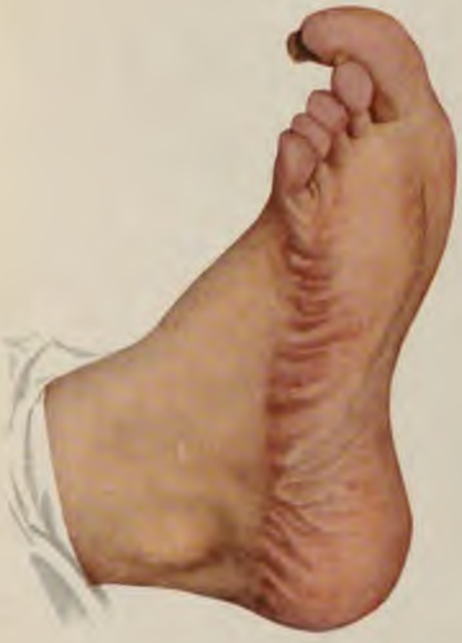
Case of **Erythromelalgia** in a man aged 52. The condition affected both lower limbs.

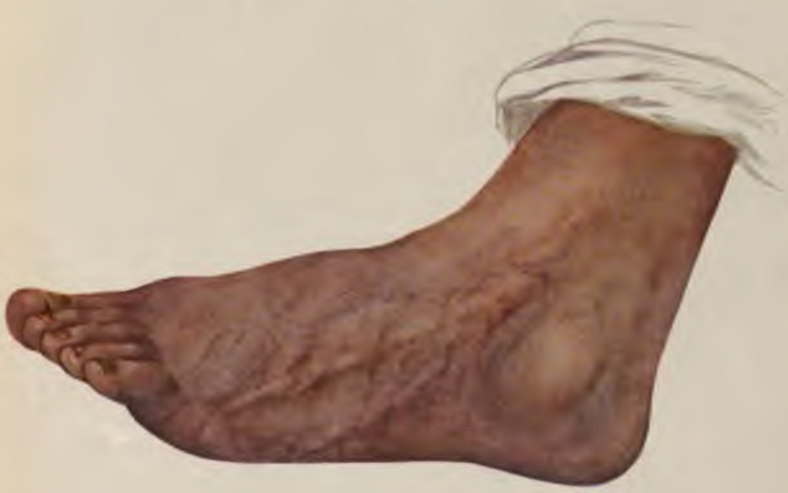
The upper drawing shows the appearance of one foot during a paroxysm when the lower limb is supported in a horizontal position. The sole of the foot is of a bright pink colour, this colour also extending on to the dorsum of the terminal phalanx of each toe.

The lower drawing shows the effect of allowing one foot to hang dependent for a few minutes. There is extreme cyanosis extending as high as the ankle. There is also severe pain, which is partially relieved by reassuming the horizontal posture of the limb.

To face page 302.







to which Bruce[1] has recently directed special attention. A few rare cases have also been observed of *family gangrene*, somewhat resembling Raynaud's disease. Fig. 164 represents three brothers, aged five, four, and three years respectively, in all of whom, one winter, areas of local necrosis appeared in the feet. The eldest child had necrosis of the skin of both heels, followed by similar areas on the dorsum of the right foot and on the fourth left toe. The second child had cyanosis and some necrosis of the dorsal surfaces of both feet, whilst the youngest had severe necrosis of the hallux, fourth and fifth toes on the right side, and a smaller area of necrosis on the plantar surface of the left hallux. It is possible that the paroxysms of *family periodic paralysis* (see p. 214) may be the result of a recurring vaso-motor spasm of the anterior spinal artery, which, as we have seen, supplies the anterior cornua of the spinal cord.

Another paroxysmal disease of angio-neurotic origin is intermittent limp, whose symptoms we have already described (p. 212), where the arteries not of the skin but of the deep structures are in a state of temporary spasm.

Acute angio-neurotic œdema is undoubtedly a vaso-neurosis. It is characterised by the sudden appearance of areas of transient œdema in the loose cellular tissue of various parts of the face, trunk, or limbs. After lasting a few hours or days, the swelling passes off spontaneously. It may also attack mucous membranes, for example, those of the respiratory or gastro-intestinal tract. A gastric attack may cause urgent vomiting, an intestinal attack meteorism, colic, and bloody diarrhœa, and if the patient happens to have an attack in the larynx, death may result from asphyxia. In the skin, the parts most often attacked are the lips, cheeks, and eyelids. The disease sometimes runs in families.

In chronic hemiplegia it is not uncommon to find œdema of the hand or foot on the paralysed side. Permanent coldness and cyanosis is very common in the paralysed limbs in cases of old anterior poliomyelitis. Even in warm weather the flaccid limb remains cold and sometimes blue in the hand or foot, as the case may be. Such cyanosis differs from that of Raynaud's group in being permanent and not paroxysmal.

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[1] *Trans. Roy. Soc. of Edin.*, 1906, vol. xlv. part i. p. 105.

Certain varieties of *urticaria* may be referable to affection of the nervous system, as in certain cases where the characteristic itching wheals appear on sudden emotional excitement. These patients appear to have a specially low coagulability of the blood due to deficiency of calcium salts in the liquor sanguinis. But urticaria is much oftener toxic in origin. *Dermographism*, on the other hand ("factitious urticaria" or "urticaria scripta"), is a reflex cutaneous phenomenon. It is elicited by stroking the skin firmly with a smooth, hard object, such as the head of a pin or the finger-nail. If, for example, we draw a diagram or write on the patient's skin in this way, a red area appears within a few seconds. The skin then becomes elevated into a hard, white ridge, which can not only be felt but seen, as if the pattern or writing had been embossed on the skin (see Fig. 182, p. 333). This lasts for many minutes and passes off gradually. Unlike true urticaria, dermographism is unaccompanied by itching. The phenomenon is commonest in neuropathic people, but is not confined to them, and may sometimes be found in apparently healthy individuals.

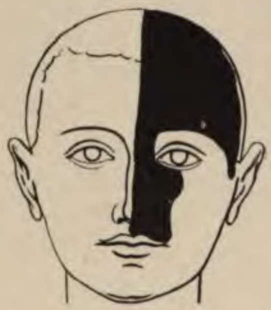

FIG. 165.—Area of localised sweating during mastication of pungent substances. From a man aged 29. The condition was congenital.

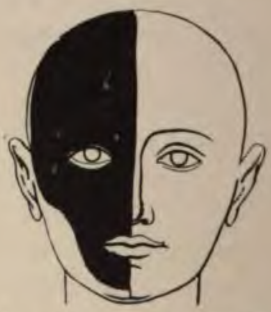

FIG. 166.—Area of localised sweating during mastication of pungent substances. From a man aged 35, in whom the condition appeared at the age of 28.

Lastly, we have to refer to certain abnormalities of sweating which are due to nervous disorders. We have already alluded to localised anidrosis or absence of sweat in certain cases of cervical sympathetic palsy. But sometimes we meet with *paroxysmal localised hyperidrosis* or excessive sweating. Figs. 165 and 166 show the areas of excessive sweating in two patients. In one the condition was congenital, and consisted in excessive sweating on the left side of the scalp and face, corresponding to the whole of the first division and part of the second division of the trigeminal.

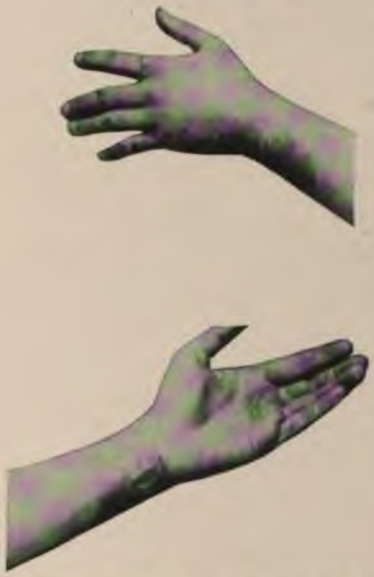

FIG. 167.—Localised hyperidrosis along ulnar border of forearm and hand. The dark area is due to powdered charcoal adhering to sweating skin.

In the other, the whole area of the trigeminal on the right side was affected and the condition appeared at the age of twenty-eight. In both these patients, who were otherwise healthy, the paroxysms of sweating occurred only on chewing highly-flavoured articles, such as onions or pickles. This condition suggests some hyper-excitability of the reflex sweating centre for the face, possibly within the pons. Fig. 167 is a photograph of an area of hyperidrosis on the left hand and wrist of a young woman of nineteen. In her, the paroxysms occurred three or four times a day, spontaneously, chiefly about the time of the menstrual period or when excited. In this case the distribution suggested a segmental or root area, apparently about the eighth cervical and first dorsal segments, and counter-irritation over the lower part of the neck, front and back, near the exit of these roots, produced rapid amelioration of the condition.

In certain chronic cases of hemiplegia, we not infrequently observe excessive sweating of the hand and foot on the paralysed side. This is doubtless due to changes in the sweating centres in the central nervous system.

LECTURE XXI

HYSTERIA

When as physicians we talk of hysteria we all have a fairly clear general idea of what we mean. And yet if we are asked the question, " What is hysteria ? " we find it somewhat difficult to express our definition in words.

At the outset we should recognise that hysteria is a real disease, as real as smallpox or cancer, and that it has a physical basis, probably of a chemical nature, although this is yet very imperfectly understood. We must draw a sharp distinction between a hysterical patient and a person who is deliberately shamming or malingering. The imitation of other diseases which is so often seen in hysteria—*neuromimesis*—is not a voluntary imitation. On the contrary, the various paralyses and pains from which so many hysterics suffer are as real to the patient as if they were due to gross organic causes.

All modern observers agree that the site of the disease is in the nervous system, and various hypotheses—vasomotor, nutritional and others—have been suggested to account for it. I believe that hysteria is a disease of nerve-metabolism affecting not only the highest or psychical centres, but implicating the whole nervous system, cerebro-spinal and sympathetic, in varying degrees, and producing symptoms corresponding with the parts affected. Although hysteria often follows some mental or physical shock, it is probable that in many cases there has been an antecedent predisposition to the disease, in the form of an unstable nervous system. But this is not always so.

Some hysterical symptoms are psychical, due to affection of the highest centres ; others, sensory, motor and reflex, are the result of affection of the sensori-motor cerebro-spinal paths ; others, again—visceral, vasomotor, &c.—are referable to the sympathetic nervous system.

Such disordered nerve-metabolism may show itself in three ways; by excessive activity, by diminished activity, or by perversion of activity.

The basis of hysteria, as of all diseases, bodily or mental, is physical in nature. In many instances this physical basis is a toxic one, the poison either being produced within the body or introduced from outside. In others, gross anatomical changes may induce hysterical symptoms. Just as gastric symptoms may result not only from faulty chemistry within the stomach but from gross organic disease in its neighbourhood, so also certain cases of serious organic disease, *e.g.* disseminated sclerosis and some cerebral tumours, may produce at first symptoms which are indistinguishable from those of hysteria—which, in fact, are hysterical. And in such cases we must try to detect the underlying organic disease and to differentiate its symptoms from those of the superadded hysteria. Hysteria, therefore, may coexist with organic disease. In practice, however, we generally restrict the term hysteria to those cases where no organic disease is present.

Let us briefly consider some of the most characteristic diagnostic symptoms of hysteria and note in what respects they differ, if they differ at all, from the analogous symptoms produced by gross organic lesions.

Space does not permit us to discuss the various predisposing and exciting causes of hysteria, such as sex, age, nervous heredity, physical and mental shock, and so on, or its frequency in certain races, especially the Latin races and the Jews.

For purposes of convenience we shall consider the symptoms in four groups—psychical, sensory, motor, and lastly, visceral and vascular.

In each of these groups we may find excess, diminution, or perversion of the normal nervous processes.

Psychical Symptoms.—These are invariably present in hysteria to a greater or less degree. The most outstanding feature is *deficiency of inhibition.* The patient reacts too readily to stimuli or suggestions, whether originating in the outside world or within her own body. One of the most striking instances of this is found in the phenomena of hypnotic suggestion, whereby a peculiar form of temporary hysteria—the hypnotic trance—is artificially induced by suggestion, and can be made suddenly to disappear by the same means. This disappearance of symptoms during or after hypnosis is sometimes taken advantage of in the treatment of hysteria by hypnotic suggestion. But it is open to the objection that instead of strengthening the patient's feeble inhibition, it utilises that fault. Yet if the result be that the patient has a hysterical suppression of her hysterical symptoms (on the mathematical principle that — × — = +) it is, to some extent, a benefit. But results obtained by this plan are less likely to be permanent than when we strengthen the patient's inhibition or self-control by positive measures.

Deficient inhibition being the keynote of the hysterical "ψυχή," we find, accordingly, that the patient is excessively emotional and changeable in disposition, often excitable and perhaps passionate. She giggles or cries on slight provocation, and one of the most familiar forms of hysterical "fit" consists merely in alternate loud laughter and crying. Her will-power is feeble, she is swayed by passing whims; and this want of self-reliance leads to another very characteristic symptom—the craving for sympathy. If that sympathy be shown, as is so often the case, to an injudicious extent by the patient's relatives and friends, her recovery may be indefinitely delayed. Hence it is generally of supreme importance to remove the hysterical patient completely from her old surroundings, and to isolate her until the vicious circle is broken.

Figs. 168 and 169 are photographs of a woman aged 37, who for over fourteen years had lain on a bed of sickness unable to move her legs, a case of hysterical paraplegia. But she was cherished by the sympathy of a devoted mother and of various benevolent lady-visitors, anxious to soothe her dying moments. She was admitted to hospital, and as a result of six weeks of isolation and massage, she recovered the power of walking, as will be seen from the other photograph (Fig. 170). And this successful result was due quite as much to the isolation as to the other measures such as special diet, and massage.

It is convenient to mention here, in connection with the psychical symptoms, the *hysterical affections of speech.* Sometimes there is excessive volubility—a *diarrhœa verborum.* In other cases we find the reverse condition of hysterical mutism, where the patient is absolutely dumb. In less severe cases it is not uncommon to find merely loss of voice, or hysterical aphonia—where the patient can only utter her tale of woe in a whisper, breathing it softly into the ear of a sympathetic listener. Hysterical aphonia has characteristic laryngoscopic appearances, in the form of adductor paralysis of the cords, with which we are familiar.

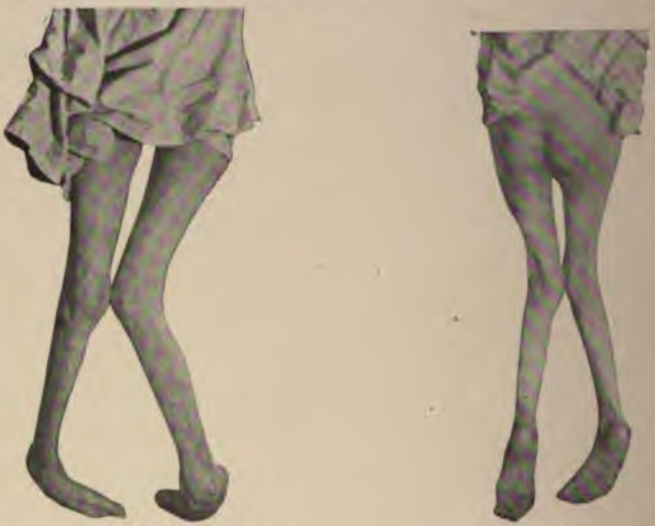

FIG. 168. FIG. 169.

Figs. 168 and 169.—Case of hysterical paraplegia of fourteen years' duration. Showing a characteristic hysterical posture of the feet when the patient is passively supported.

Patients with mutism or aphonia sometimes suddenly recover their voice when the dominant obsession is loosened, *e.g.* by administration of chloroform (by the physician), or of an excess of alcohol (by the patient), or by some sudden shock, physical or mental. Intra-laryngeal faradism cures immediately many cases of hysterical aphonia.

Articulation in hysterical patients may be affected in all sorts of curious fashions. I have seen several patients who drew a breath between each separate syllable, *e.g.* "hos—pi—tal." Others interpolate curious barking, grunting, or snorting noises amongst their words. One lady was afflicted in this way to such an extent that new neighbours who settled in an adjoining house

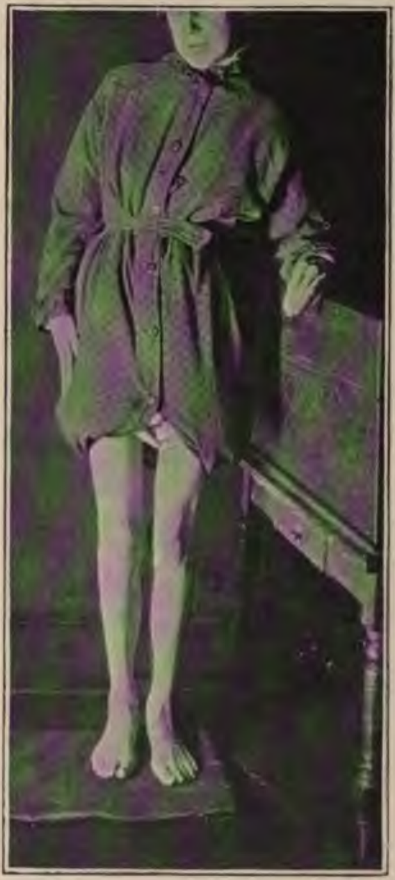

FIG. 170.—The same patient as in Figs. 168 and 169, after six weeks' treatment, showing restoration of power of walking.

thought the noise was made by a sick dog, and made a humane suggestion that the animal should be put out of its pain. And yet this lady could recite long dramatic passages of poetry and prose, though in ordinary conversation, or even when not talking, her bark made her society a mixed pleasure.

Stammering in its different varieties is not uncommon in hysterical patients. Unlike ordinary stammering which comes on in childhood, hysterical stammering may develope suddenly in adult life. Thus in a hospital nurse aged 33, who stood up suddenly and knocked her head against a mantel-shelf, severe hysterical hemiplegia came on next day, and stammering some six weeks later, lasting for several months.

Sensory Symptoms.—These are of greater diagnostic importance than is commonly realised. Pain of some sort occurs in nearly every case of hysteria. Unlike ordinary pains, which are generally caused by some peripheral irritation, hysterical pains are entirely central in origin—*psychalgia*—and should really be classed as hallucinations. But in every case we must be careful to see and to examine the painful spot, and to exclude peripheral irritation, before labelling any pain as hysterical.

Hysterical pains may be referred to any part of the body; but they are especially common in certain situations. For example hysterical headache is often of the "clavus" type, which is a boring pain localised to one small spot on the skull. Occipital headache is particularly common, so is hemicrania. Pains in the spine may simulate those of organic disease. Pain in the breast —mastodynia—or in the joints may lead to difficulties in diagnosis. It is only by careful local and general examination, revealing the presence of other hysterical stigmata and the absence of signs of structural disease, that we can avoid errors. Cases have been known in which amputations have been performed for hysterical joint-pains. Some time ago I saw a young lady who had already had one toe removed by a surgeon, but the pain recurred in another toe, and the case was undoubtedly hysterical.

Closely related to these pains are the *areas of hysterical hyperæsthesia*, and especially of hyperalgesia or excessive tenderness.

Universal hyperæsthesia is rare. We also meet with cases of hemi-hyperæsthesia. More usually this excessive tenderness is circumscribed in small areas—little islands of skin or subjacent tissues of the head, trunk, or limbs. Sometimes the tenderness is cutaneous and elicited by gentle stroking of the skin; sometimes it is subcutaneous, and only elicited on deeper pressure.

Such tender points are chiefly situated in the vertebral, infra-mammary, epigastric, and inguinal regions, and except when mesial, are more frequently left-sided (except in left-handed people, in whom they are more commonly right-sided). Tender points are less common on the head, and rarest on the limbs.

Of all the tender spots, that in the left inguinal region is perhaps the commonest. From some supposed connection with the ovary, it has been called "ovarian" tenderness, but the symptom is as frequent in male hysterics as in females, so that the term is a misnomer. Moreover, in this connection, Steinhausen[1] examined 500 healthy soldiers—males, not Amazons—and found that in no less than 88 per cent. brisk pressure in the inguinal region on either side produced a reaction of some sort, the phenomena being either sensory (unpleasant tickling or pain), motor (hardening of abdominal muscles, various reflex and protective movements), psychical, or vasomotor and sympathetic (dilatation of pupils). And yet there was not a single ovary amongst them.

These tender points may be associated not merely with pain, but with so much disturbance as to be actually *hysterogenic*. This does not mean that they induce hysteria—the hysteria is already present. It means that pressure on such a spot induces a hysterical fit or paroxysm. The best-known hysterogenic area is in the left inguinal region, but such areas may be anywhere. I know of one patient who had a hysterogenic spot in one axilla and who wore a sort of truss over it, to prevent accidental pressure.

Sometimes deeper pressure on the hysterogenic spot or elsewhere may arrest a hysterical fit when in progress. Areas, pressure on which causes cessation of the paroxysms, are called *hystero-*

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[1] Steinhausen, *Ueber die physiologische Grundlage der hysterischen Ovarie. Deutsche Zeitsch. f. Nervenheilk.*, xix. p. 369.

frenic. The inguinal region is the best known of these. Strong faradism over the inguinal region will stop most hysterical fits.

Hysterical hyperæsthesia may also affect the special senses, so that there may be hyper-sensitiveness of smell, vision, hearing, or taste. This is less common than loss or diminution of special senses, to which we shall refer later. I had for some time under my care a patient who could not tolerate bright light, especially if the room had a blue wall-paper. He preferred to stay in a darkened chamber; or if the blinds were up, he shaded his eyes with his hand. After some weeks of treatment, he completely lost this photophobia.

In rare cases an actual enlargement of the visual field has been observed, generally in one eye only. Thus, in a soldier with hysterical wry-neck and anæsthesia of one side of the body, the visual field on the non-anæsthetic side, when measured with the perimeter, was much larger than in a normal individual.

Hysterical anæsthesia is extremely common, and is of the greatest diagnostic value. A degree of anæsthesia exists, I am convinced, in the overwhelming majority of hysterical cases, except those occurring in childhood. Some time ago I looked through my notes of 62 consecutive cases and found that anæsthesia was present in 49 and absent only in 12. The remaining case of the series had unilateral hyperæsthesia.

Hysterical anæsthesia is often unnoticed by the patient herself, and only discovered on examination by the physician. Sometimes, however, the patient complains of actual numbness, and this is chiefly in cases where the affected limb has also motor paralysis, so that her attention is called to it.

Janet, of Paris, showed an ingenious method of demonstrating that in some cases of hysterical anæsthesia, sensory impulses really reach the brain-centres, though the patient does not consciously perceive them. Taking a case of complete hemi-anæsthesia he makes the patient shut her eyes, and tells her to say "Yes" each time she feels a touch or prick, and to say "No" when she does not feel it. In certain cases the patient not only says "Yes" every time she is touched on the normal side, but also says "No" every time she is touched upon the anæsthetic side. This is pathognomonic of hysteria.

Cutaneous anæsthesia in hysteria may be complete or partial in degree, or it may be dissociated. Diminution or loss of painful sensations is even commoner than tactile anæsthesia. Many of the mediæval witches were simply hysterics. Their hysterical analgesia was usually demonstrated by sticking pins into them, and if an analgesic area was discovered, they were promptly thrown into the nearest pond or stream. If they sank and were drowned, their innocence was established; but if they floated, it was additional evidence of guilt.

But whatever be its degree of intensity, hysterical anæsthesia never maps out an individual nerve-area such as that of the radial, median, ulnar, or external popliteal. Its commonest distribution is a *hemi-anæsthesia* (26 out of 49 cases) which is mostly left-sided, except in left-handed patients.

Hysterical hemi-anæsthesia, including the accessible mucous membranes of the eye, nose, mouth, pharynx, vagina, and rectum, is usually accurately bounded by the middle line; but not always. It may either extend farther over and encroach on the non-anæsthetic side, or it may leave certain areas with normal sensation, even on its own side, especially the head and the genitals, as may be seen from some of the charts (Figs 172 and 173).

Bilateral universal anæsthesia is rare (see Fig. 68, p. 165). We generally find, somewhere or other, one or more islands of normal sensation, or even of hyper-sensitiveness.

In many cases the anæsthesia, though unilateral, is more marked on the face or limbs than on the trunk. Or it may affect special levels of a limb (knee, elbow, or shoulder), or it may stop abruptly at some horizontal line (shoe, sock, stocking, mitten, glove, sleeve). This "*segmental*" *anæsthesia* sometimes occurs in association with hemi-anæsthesia (10 out of 49 cases) or by itself (12 out of 49) on one or both sides (Figs. 174 to 176).

The mode of onset and disappearance of hysterical anæsthesia is interesting. Sometimes the anæsthesia comes on gradually, and the patient is unconscious of the defect. In other cases it occurs

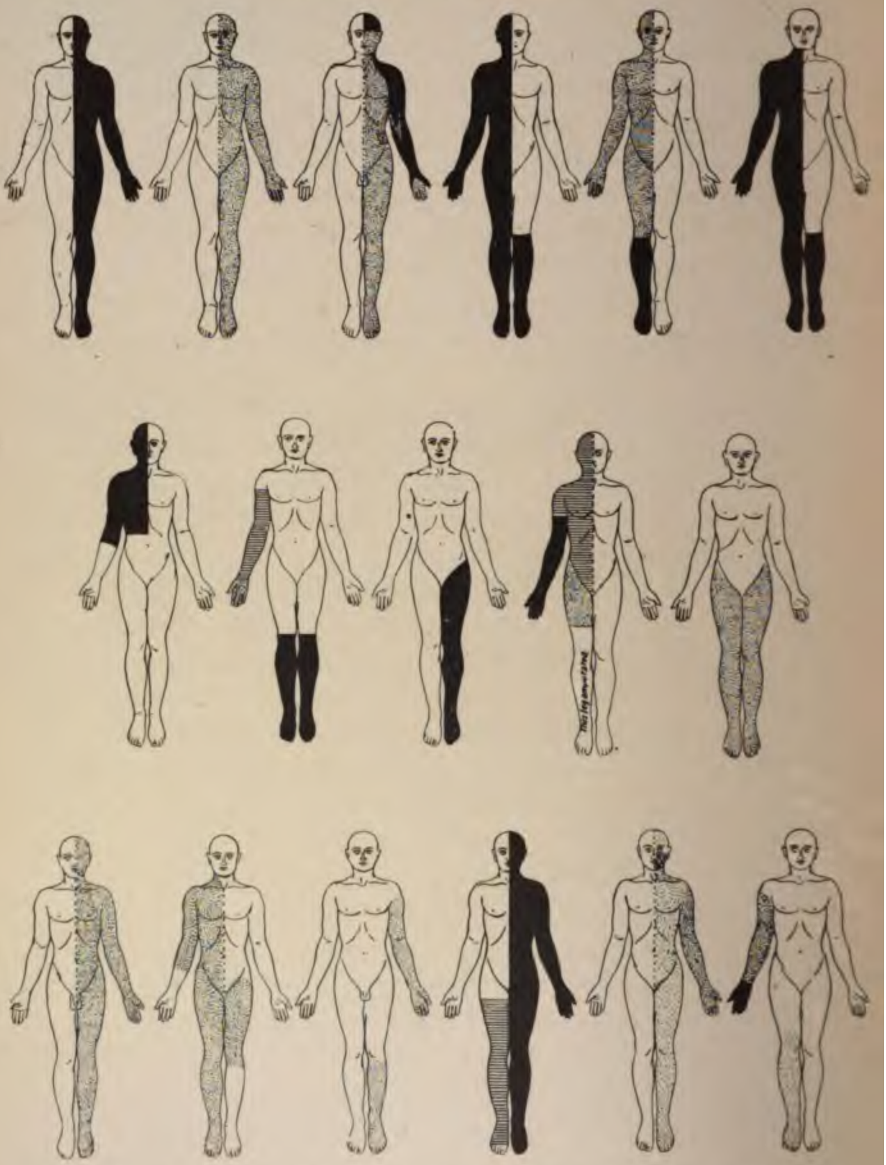


FIG. 171.—Various types of hysterical anæsthesia. Dotted areas indicate slight sensory loss: shaded areas more severe impairment, and black areas total loss of sensation.

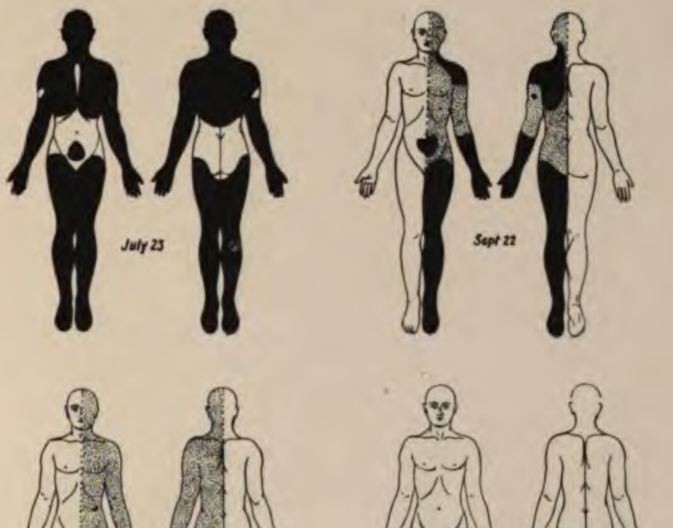


FIG. 172.—Case of hysterical paralysis in a girl aged 17, showing progressive improvement in the anæsthesia.

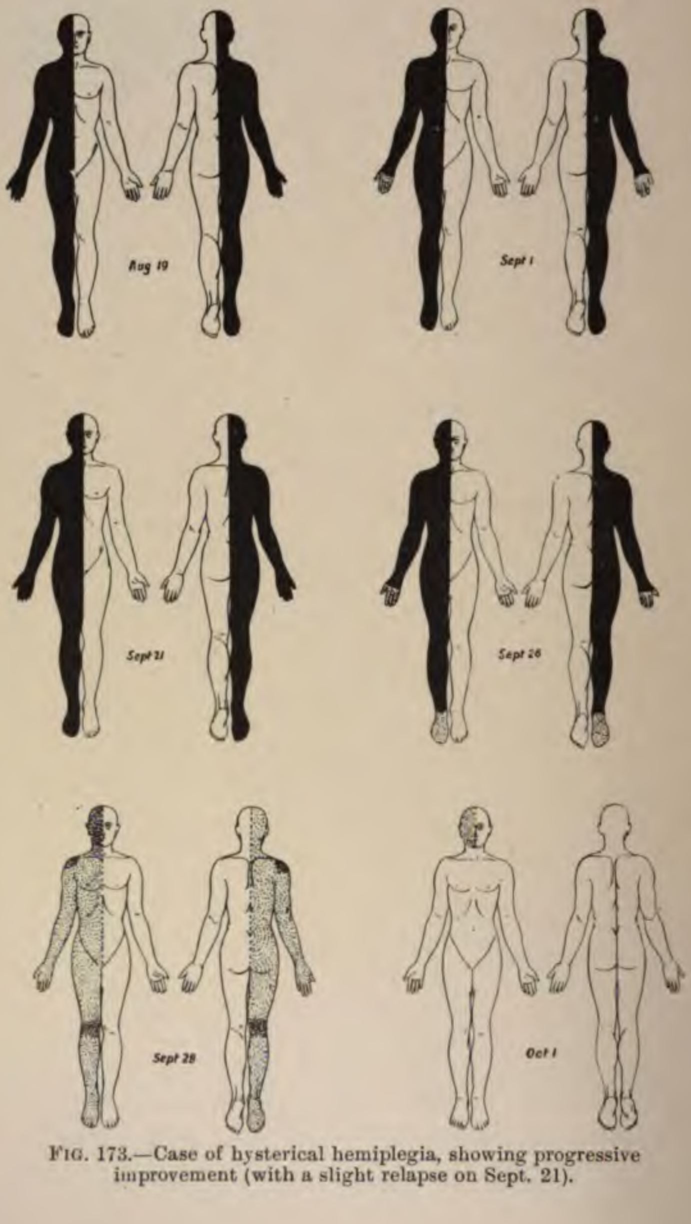


FIG. 173.—Case of hysterical hemiplegia, showing progressive improvement (with a slight relapse on Sept. 21).

suddenly, especially after a hysterical fit, and the patient is then more likely to notice her " numbness."

We seldom have the chance of watching the onset of hysterical anæsthesia, but we may often study its mode of disappearance, and Figs. 172 to 176 show charts of several cases of hysterical anæsthesia in various stages of recovery. Unlike organic anæsthesia, which, if it recovers, fades gradually all over the recovering area, hysterical hemi-anæsthesia may suddenly fade to segmental (Fig. 172), and segmental anæsthesia recedes by jumps from a higher to a lower level, bounded usually by an " amputation line " drawn transversely across the limb. Sometimes it relapses temporarily to its old level before resuming its progress towards recovery (Figs. 174 and 175). More rarely it clears up first at the periphery.

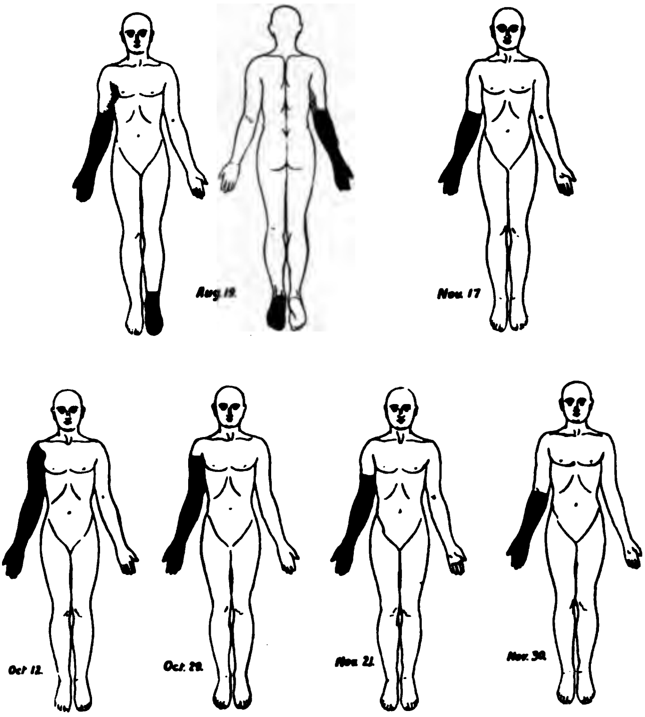


FIG. 174.—Case of hysterical paralysis in a left-handed patient, showing progressive improvement in anæsthesia.

Anæsthesia of Special Senses.—Most cases of hysterical anæsthesia also have diminution or loss of the special senses—smell, vision, taste, and hearing—generally unilateral and on the same side as the cutaneous anæsthesia, rarely on the opposite side. This combination of unilateral affection of special senses and of cutaneous sensation is pathognomonic of hysteria, and does not occur in organic disease.

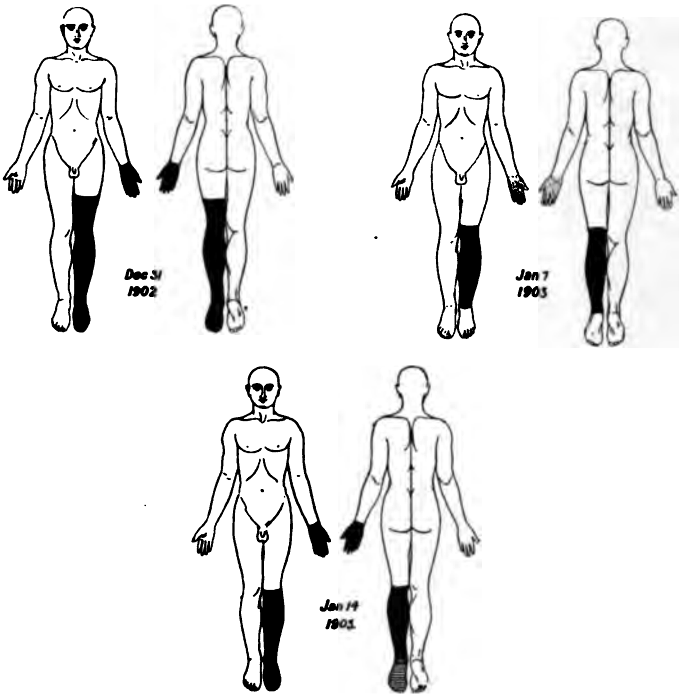


FIG. 175.—Case of hysterical hemiplegia, showing variations in anæsthesia.

The affection of vision in hysteria is not a hemianopia such as we often get in organic hemiplegia. It is a concentric contraction of the whole visual field, as will be seen from the accompanying perimetric charts (Fig. 177). It is more marked in one eye than in the other—"crossed amblyopia"—the smaller field being on the hemi-anæsthetic side, usually the left.

The colour-fields are also contracted, but in a different order to that which occurs in organic optic atrophy. In hysteria the blue field diminishes first and the red last of all, whereas in organic atrophy the red field is usually first affected.

Sometimes, when charting the visual field of a hysterical patient. we notice that the field becomes progressively smaller and smaller, as we continue our examination, so that our perimetric outline has a *helicoid* or spiral shape (Fig. 179). This form of perimetric tracing does not occur in organic disease.

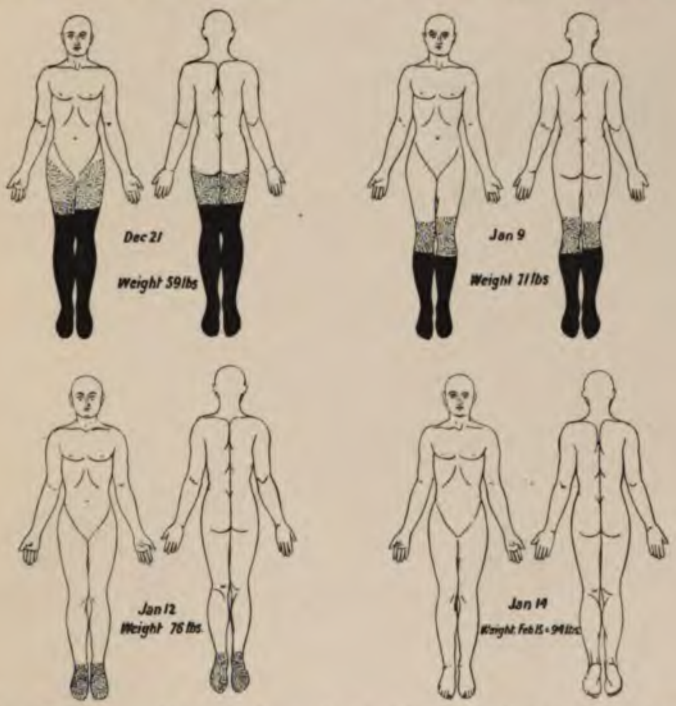
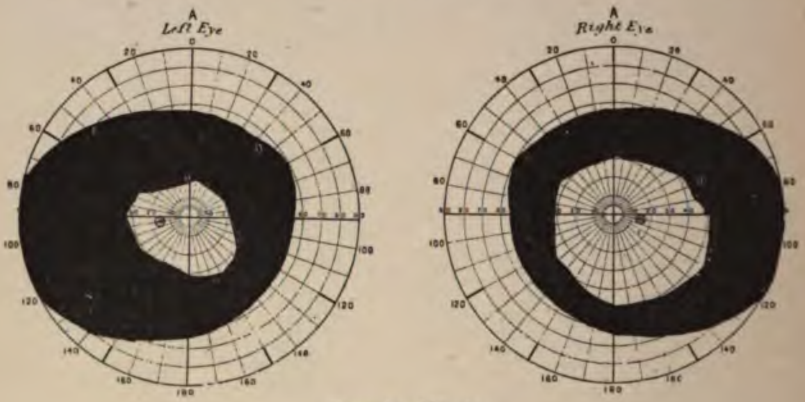


FIG. 176.—Case of hysterical paraplegia with anæthesia in a girl aged 19, showing progressive improvement.

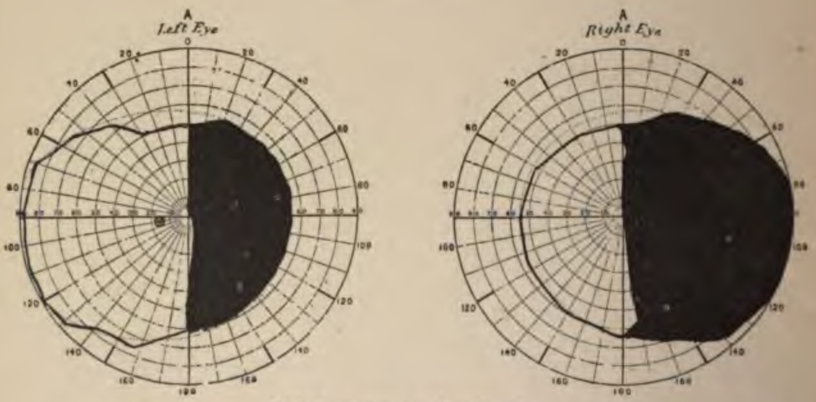
Sometimes we have actual hysterical blindness or amaurosis in one eye, and yet by means of prisms we may produce a diplopia, which in organic cases would be impossible. In very rare cases complete bilateral hysterical blindness has been observed.

Time does not permit us to discuss the other peculiarities of hysterical eye-affections and of hysterical loss of smell, taste, and hearing, but we may mention, in passing, that complete bilateral loss of taste, as Hughlings Jackson has pointed out, is practically always hysterical.

Hysterical Paræsthesia, or Perversion of Sensation.—In some cases of hysteria a touch on one limb or one side of the body is felt by the patient at the corresponding spot on the opposite side—*allocheiria* (Obersteiner). In another variety, of which I have seen an example, a touch on the radial border of the limb was felt on the ulnar, and *vice versâ*. *Haphalgesia* (Pitres) is the term used



Crossed amblyopia.



Right homonymous hemianopia.

FIG. 177.—Visual fields from cases of hysterical and organic hemiplegia respectively, in which vision was affected.

when intense pain is caused by touching the patient with certain substances, such as metals, which normally should only cause a tactile sensation.

Paræsthesia may also affect the special senses, as for example in *monocular diplopia* or *polyopia*, which is always hysterical, and in *micropsia*, when everything looks very minute, or *macropsia*, when surrounding objects seem gigantic.

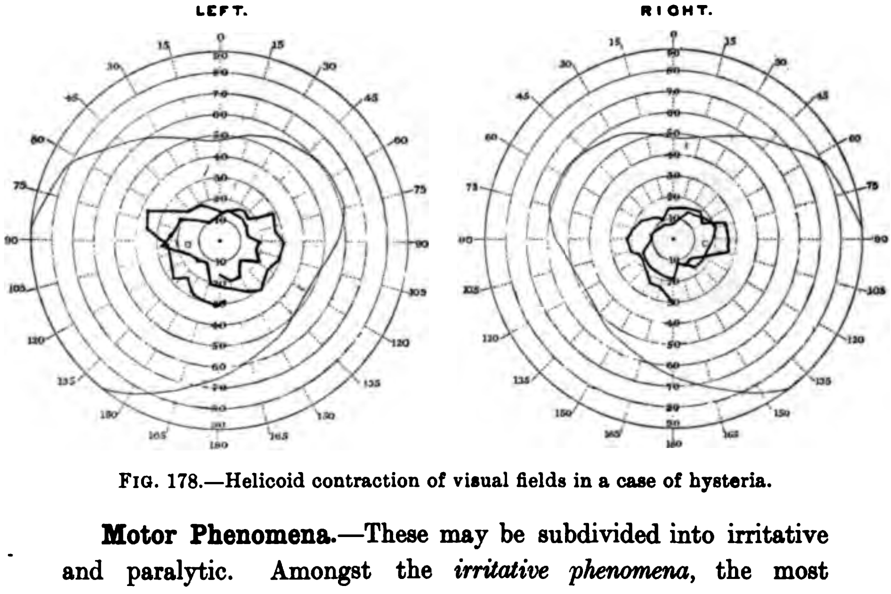


FIG. 178.—Helicoid contraction of visual fields in a case of hysteria.

Motor Phenomena.—These may be subdivided into irritative and paralytic. Amongst the *irritative phenomena*, the most striking are the so-called hysterical fits or paroxysms. Hysterical fits vary enormously in type and in severity, from a simple emotional outburst of uncontrollable laughing or crying, accompanied perhaps by the hysterical " globus " or " ball in the throat," to the most prolonged, dramatic, violent muscular movements, together with apparent unconsciousness.

One variety of hysterical fit is sometimes mistaken for epilepsy, and there is all the greater liability to make this mistake since the fit is generally over before we reach the patient, and we are dependent for our information upon the accounts, more or less accurate, of unskilled witnesses. But if the physician is lucky enough to be present during a fit, there is seldom any difficulty in diagnosis. Thus, for example, the patient never hurts herself in falling; there is no stertorous breathing; her face is not livid, nor does she bite her tongue as in epilepsy; but she may bite her lips or snap at the fingers of the bystanders. She never empties the bladder or rectum during the fit; her eyes are generally tightly closed, and if the physician tries to open them, the patient actively resists. A hysterical fit is not followed by coma and hardly ever by vomiting.

But we must remember that sometimes we have hysterical fits which are post-epileptic—*i.e.* which immediately succeed an attack of true epilepsy. The antecedent epileptic fit in such cases is usually of the "petit-mal" type, consisting perhaps in a mere transient pallor of the face, with momentary loss of consciousness, and then passing directly into a hysterical fit. Therefore, in every case it is important to inquire very carefully as to the precise mode of onset, lest we overlook a case of combined epilepsy and hysteria.

Charcot's "*grande hystérie*," with its initial period simulating epilepsy and its subsequent phases of contortions, kicking, and struggling (clownism), passionate attitudes (opisthotonos, crucifixion attitude, &c.), and delirium, often with hallucinations of animals, is less common in this country than in France, but once seen, it is a magnificent performance and can never be forgotten. It is totally unlike any kind of epileptic or organic fit, and its diagnosis is easy. It may last from a quarter of an hour to several hours at a time. This "status hystericus" is commoner than the "status epilepticus," but the patient has no subsequent stupor such as that which succeeds a severe epileptic fit.

Amongst other varieties of fits, to which we can only briefly refer, we may mention *catalepsy*, in which the patient suddenly becomes speechless, motionless, and stiff. Sometimes she is conscious all through the fit, at other times she is in a dreamy mental state. Meanwhile, if the limbs be passively moved into any posture, however fastastic, they remain fixed there like a doll's limbs. In one case of mine the patient, a woman of 28, could be lifted up during the attack by the head and heels and laid across two chairs like a log.

Hysterical trance may come on spontaneously, or may succeed a hysterical paroxysm. It is a condition in which the patient appears as if in a deep sleep; but the muscles are seldom completely relaxed, and we may observe slight tremors of the eyelids. In more severe cases, the heart and respiration may become so feeble and slow that the condition simulates death. Hysterical trance may last hours, days, or weeks; and several cases have been authenticated where such a patient has been buried alive, either deliberately, as in the case of some Indian fakirs, or by accident in this country. Novelists know this, and when the heroine is thus buried, she is exhumed in the last chapter by the villain for the sake of a magnificent diamond ring on her finger; whilst her finger is being cut, to get the ring off, the patient awakes.

We can only mention other varieties, such as *somnambulism* and *double consciousness*, this latter being a condition in which the patient's character alternates between normal and abnormal, the two individualities being mutually unconscious of each other, but each one, as its turn comes, takes up the thread where it left off last.

Various *localised spasms* also occur in hysteria. Such, for example, is the large group of "tics" and "habit spasms" which we have already studied (p. 75). A true tic is essentially and primarily a psycho-motor act, either an emotional expression or a movement which has become a habit. Of these tics, the commonest are grimaces, jerkings of the head, trunk, or limbs, and tremors of various parts, rapid or slow. Thus, for example, a lady's maid, aged 46, had tonic spasm of the orbicularis oculi on both sides (blepharospasm), and could open her eyes only by opening the mouth as well. Another girl, æt. 19, had a clonic or jerking blepharospasm, paroxysmal and associated with "humping up" of one foot. Another girl of 19 had rapid "twiddling" movements of the left thumb and fingers, with pronation-supination movements of the forearm and a pseudo-clonus of the left ankle. Another girl aged 20, who had suffered long previously from recurrent vulval abscesses, had attacks of rapid antero-posterior movements of the pelvis. Another patient had rapid violent flexion-extension movements of the left elbow whenever a thunderstorm occurred, the hand meanwhile dangling loosely at the wrist. Such cases of localised spasms might be multiplied almost *ad infinitum*.

Hysterical Paralysis may be either flaccid or spastic in type, and may affect any of the voluntary muscles; but, unlike paralysis due to organic lesions, it never attacks a single muscle nor the muscles supplied by a single nerve, nor are the electrical reactions of degeneration ever present. And a further point about hysterical paralysis is that though it may roughly resemble the posture of an organic paralysis, it never does so with accuracy; there is always some point of difference to be detected. The reason for this we have already discussed (p. 231).

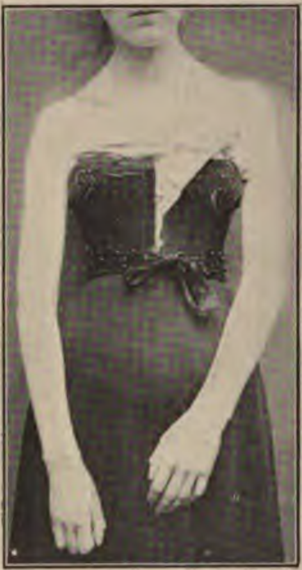


FIG. 179.—Hysterical monoplegia of right upper limb, accompanied by muscular atrophy, but without changes in electrical reactions.

Let us study one example each of hysterical monoplegia, of paraplegia, and of hemiplegia.

Fig. 179 is that of a nurse, aged 32, with hysterical monoplegia of the right arm of eight months' duration, in whom there was extreme muscular wasting and claw-hand. The paralysis came on after a strain of the shoulder in lifting a heavy patient, and somewhat resembled a lesion of the brachial plexus. But we observed that the trapezius was paralysed, and that the whole scapula was displaced downwards—unlike an organic brachial-plexus case, in which the arm would be displaced downwards at the shoulder-joint. Moreover, the electrical reactions were normal, and there was a "glove" of anæsthesia, unlike the "root" anæsthesia of an organic case. The patient was treated for several weeks by battery and massage, without effect at first, but the result proved our diagnosis to be correct, for at a religious meeting she was suddenly cured.

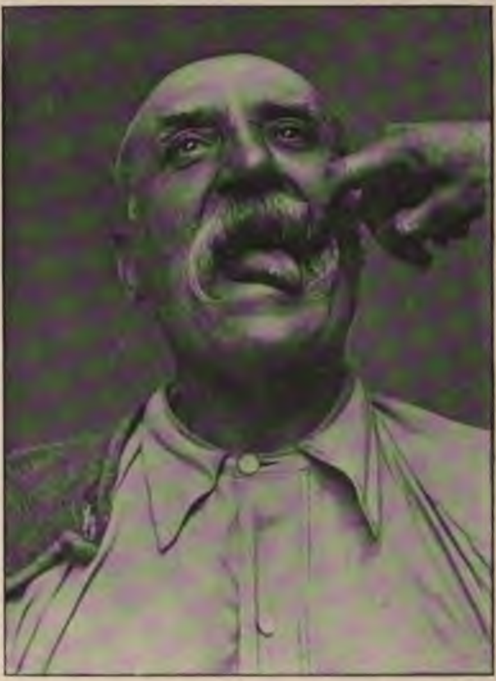


FIG. 180.—Left-sided hemi-glossic spasm in a case of hysterical hemiplegia, showing position of tongue when protruded.

Figs. 168 and 169 are from a case of hysterical flaccid paraplegia of fourteen years' duration in a woman of 37, which at first sight might be mistaken for a cord-lesion with muscular atrophy and anæsthesia. But the anæsthesia was of the "stocking" type, the electrical reactions were normal, the sphincters were unaffected, and there were no bed-sores; and the result of six weeks' isolation and massage was to restore the power of walking (Fig. 170).

In some cases of hemiplegia the posture is sufficient to diagnose hysteria. For example, in the patient shown in Figs. 106 and 107 (p. 231), instead of the ordinary posture of an organic case, with the upper extremity flexed and pronated, the lower extremity flexed at the hip, extended at the knee and ankle, and slightly inverted, there was a curious posture of the hand and foot unlike that of organic disease. Moreover, her face entirely escaped, in spite of the severe paralysis of the arm and leg; and she had hemi-anæsthesia and loss of special senses down one side, a combination which never occurs in organic hemiplegia.

The face and tongue are rarely affected in hysterical hemiplegia. But in rare cases we may find instead of weakness, spasm of the face and tongue on the affected side, when the patient shows the teeth or protrudes the tongue. This hysterical *glosso-labial hemi-spasm* is exceedingly rare, but Figs. 180 and 109 (p. 232) are good examples of the condition.

We may also refer to "Lasègue's symptom," in which a patient with an anæsthetic limb cannot move it when the eyes are closed, but is able to move it when she opens her eyes and looks at it. Another feature worthy of mention is hysterical pseudo-ptosis. In organic ptosis due to lesion of the third cranial nerve, there is always a compensatory over-action of the frontalis muscle. But in hysterical pseudo-ptosis this is absent, and the condition is really due, not to paralysis of the levator palpebræ, but to spasm of the orbicularis oculi (see Fig. 45, p. 110).

Before leaving the motor phenomena of hysteria, it may be well to refer to the gaits of hysterical patients, which are sometimes most peculiar. Thus one hysteric, a doctor aged 55, used to flourish his left leg in the air and bring it down with a stamp like that of a unilateral locomotor ataxia. The phenomenon was so dramatic that he had to carry a stick to beat off the crowds of little boys who studied his gait in the streets.

Astasia-abasia is a hysterical condition in which, although the patient can move his legs normally when lying or sitting, he collapses at once when he tries to stand or walk. A boy, aged 12, had this symptom for a long time, but was cured by an application of the wire-brush to his spine. Another patient was a worthy married lady who every now and then, when walking, sat down suddenly on the ground, rolled backwards and spread out her lower limbs like the letter V.

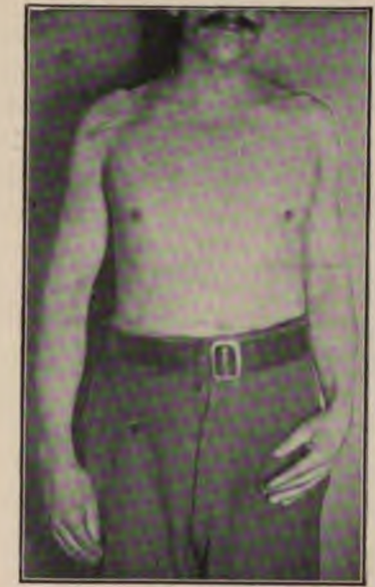


FIG. 181.—Hysterical contracture of the left hand and elbow, following an injury to the elbow.

Perhaps the commonest hysterical gait is a dragging gait, in which the patient trails the limb helplessly along, often scraping the inner border, or even the dorsum of the foot, on the ground (see Fig. 116, p. 239), unlike an organic hemiplegia in which the outer side of the sole is dragged.

Some authorities state that muscular atrophy does not occur in a hysterical limb. But this is not quite accurate, for in certain rare cases, one of which is shown in Fig. 179, we may meet with advanced atrophy. But this atrophy is due to disuse and is not associated with the electrical reactions of degeneration.

Contractures of the most pronounced type may be met with in hysterical paralysis. But here, again, they always differ in some respect from those of organic cases. Fig. 181 is the photograph of a soldier, aged 32, whose horse rolled on him at the Tugela, and who afterwards developed a stiff arm, flexed at the elbow and wrist, and with the thumb and index finger held stiffly parallel.

A study of the **reflexes** is of great diagnostic importance in every case of hysteria. The deep reflexes may be normal or exaggerated, but in pure hysteria the knee-jerks are never lost. True ankle-clonus does not occur, but a pseudo-ankle-clonus is often met with. In a girl of 19, the subject of thread-worms and pruritus ani, this pseudo-ankle-clonus used to come on spontaneously when sitting or standing at ease. One can often distinguish it from a true organic clonus by the peculiar upward start of the foot before it sets off on its first downward push. Also pseudo-clonus is, as a rule, poorly sustained.

The superficial reflexes are often diminished or absent, especially on the anæsthetic side. The plantar reflex in hysteria, if present, is always of the normal flexor type, never of the extensor or Babinski type. An extensor plantar reflex practically only occurs in cases of disease of the pyramidal tracts, and in infants who have not learned to walk and in whom the pyramidal tracts are not yet developed.

The pupil-reflex to light is never lost in pure hysteria, though in rare cases it may be "concealed" by the presence of pupillary spasm. I remember one case of fixed dilated pupils in a hysterical woman, but this was due to the taking of belladonna by the patient herself.

As to the bladder and rectum, although we may have frequency of micturition in hysteria, we never have true incontinency. Retention of urine, on the other hand, is a fairly common symptom. It once broke out as an acute epidemic in a school for young ladies and continued until the doctor judiciously handed over the duty of catherisation to a female nurse of mature years. The symptom at once subsided in a gratifying manner.

Visceral and Vasomotor Phenomena.—It is important to remember that hysteria affects the sympathetic nervous system as well as the cerebro-spinal. Let us refer very briefly to some of the visceral and vascular phenomena.

We may meet with abnormal slowness of the heart, or we may observe abnormal rapidity with palpitation, chiefly paroxysmal and constituting a variety of pseudo-angina, especially in hysterical young mothers who have been lactating too long. These cases are easily distinguished from true angina by the absence of signs of organic cardio-vascular disease.

In the digestive system we meet with curious hysterical phenomena. Aërophagy, or swallowing of air, is achieved chiefly by gulping movements of the pharynx. I remember a little schoolboy who could swallow air and distend his abdomen till his waistcoat could not be buttoned. We are all familiar with hysterical dysphagia or spasm of the œsophagus, with its sudden intermissions and the difficulty experienced equally with liquids and with solids, in which nevertheless a large stomach-tube can be easily passed. The hysterical "globus" or "ball in the throat," which the patient tries to swallow, is an emotional phenomenon often met with at the onset of a hysterical paroxysm. Hysterical vomiting has always to be excluded in gastric disorders of young women. It is often associated with *anorexia nervosa*, where the patient will take hardly any food. "Fasting girls," of whom we now and then read in the daily press, are generally examples of this kind of hysteria. And they may become extraordinarily emaciated, but even they do take a little food now and then.

Rhythmic movements of the stomach or intestine with curious rumbling noises are sometimes met with. The commonest variety is the intestinal, and one sometimes hears these noises at dinner-parties in nervous young servant-maids waiting at table. Much less frequently we hear violent gastric borborygmi. One young girl whom I saw had constant, noisy to-and-fro gurgling in the upper part of the abdomen, like a steam-pump, and on palpating the abdomen the stomach could be felt rhythmically contracting and relaxing, blowing and sucking air backwards and forwards through the pylorus. This phenomenon was so startling to strangers that the unfortunate girl had to retire to her own room, if friends came to call on the family. Somewhat similar abdominal noises in another hysterical patient, a lad of 19, were apparently produced by spasmodic contractions of the diaphragm, for they ceased when he drew a deep breath and held it.

The French patient who earned an honest living at a Parisian music hall by making musical noises with his anus was probably another example of visceral hysteria.

We must also bear in mind the pseudo-pregnancies which now and then occur, in which spurious enlargement of the abdomen sometimes goes on to a spurious labour. Then " parturiunt montes, nascitur ridiculus mus "—all that is produced being, at the most, a small uterine cast. Phantom abdominal tumours can best be differentiated from genuine ovarian or uterine enlargements by giving an anæsthetic, when the abdomen at once collapses.

It is sometimes more difficult to diagnose pseudo-appendicitis. Thus one patient whom I saw, aged 33, had had her abdomen opened twice in different London hospitals for supposed appendicitis, the symptoms being those of recurrent pain and tenderness in the right iliac fossa, with constipation and vomiting. But she had also right-sided hemi-anæsthesia, with loss of special senses all down that side. We were thus led to suspect the hysterical nature of the abdominal symptoms, and accordingly her next attack was cured by sal volatile, without laparotomy, and she has had none since.

Hysterical diarrhœa sometimes occurs, as in the case of a public speaker who was often attacked in this awkward way just when his turn arrived to address the audience.

Spontaneous hæmorrhages are very rare in hysteria, and no hæmorrhage should ever be diagnosed as hysterical unless all other causes can be excluded. But a certain number of cases of pseudo-hæmoptysis and pseudo-hæmatemesis occur. In one girl whom I watched, the phenomenon seemed to be produced by sucking of the gums; in another it was apparently the result of pharyngeal suction. In both cases, physicians of wide experience who saw the case in consultation failed to discover any organic cause in the chest or abdomen.

Sometimes a limb affected by hysterical paralysis and anæsthesia may show abnormal vasomotor spasm, so that if pricked or cut it bleeds less freely than normal.

Secretory phenomena also occur, though rarely, as for example in blood-stained tears or blood-stained sweat, which may be unilateral. Polyuria often occurs after a hysterical fit, whereas hysterical anuria or suppression of urine is extremely uncommon.

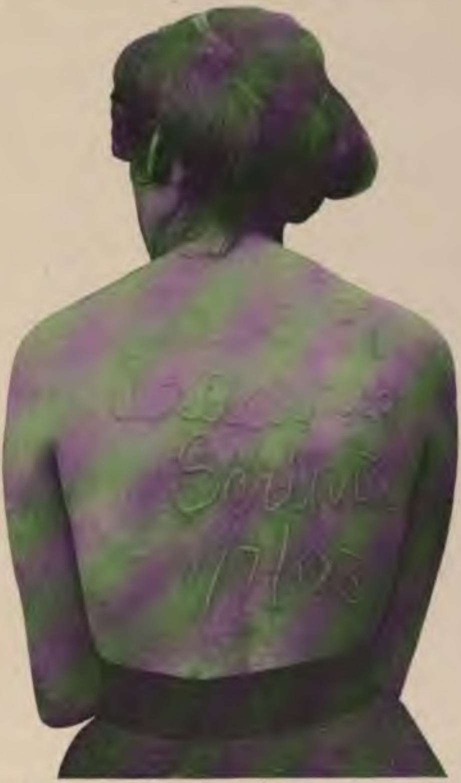


FIG. 182.—Case of dermographism in a hysterical young woman.

Certain skin affections occur in hysteria. Cutaneous hæmorrhages are rare, if we exclude cases of voluntary traumatism. Bedsores do not occur. The gangrenous patches described as hysterical gangrene are always self-inflicted, by means of caustics or other methods. Hysterical blue œdema sometimes occurs, especially in contractured limbs; it usually affects the skin over a joint and produces a degree of cyanosis and swelling, but this swelling does not pit on pressure. Dermographism is commoner in hysterics than in normal people. Fig. 182 is an excellent example in a girl with hysterical tremor of the legs in whom, when the skin was stroked with the finger-nail, a white raised wheal appeared and remained for an hour or more. This "factitious urticaria," or "urticaria scripta," unlike ordinary urticaria, does not itch.

Hysterical cough is very common; it is usually loud and hacking, going on all day and ceasing during sleep. It is not accompanied by expectoration, and is commonest in young hysterics. We may have other peculiar modifications of respiration. Thus in a girl of 24 who had hysterical fits, expiration was a curious grunting noise of a bigeminal type—two grunts between each inspiration. We also meet with paroxysmal rapid breathing, sneezing, hiccup, and yawning. One girl aged 10, the subject of hysterical hemi-anæsthesia, yawned persistently for three weeks during her waking hours. She then stopped and had an attack of hysterical mutism lasting for two months.

When hysteria occurs in childhood it is often monosymptomatic and the ordinary hysterical stigmata are frequently absent. Girls are much more frequently affected than boys, even before the age of puberty. Perhaps the commonest symptoms of hysteria in childhood are astasia-abasia, hysterical aphonia, and the various forms of habit-spasm.

The diagnosis between hysteria and organic disease is sometimes easy; in other cases it is a matter of extreme difficulty. In doubtful cases special attention should be paid not only to the psychical symptoms but to the special senses, to the condition of the optic discs, to the type of anæsthesia which may be present, to the posture of the limbs in cases with motor paralysis, and to the condition of the reflexes, especially the plantar reflex and the bladder functions. And we should never forget that hysteria and organic disease may coexist in the same case.

LECTURE XXII

ELECTRO-DIAGNOSIS AND ELECTRO-PROGNOSIS

It is not necessary to enter into a full discussion of the various physiological phenomena produced by electrical stimulation of different tissues, still less to discuss the nature of electricity itself or the *rationale* of its effects. It will suffice here to recall a few of the more practical points in the physiology of electrical stimulation.

Clinically, electrical stimulation is of value chiefly in the examination of muscles and of motor nerves. Electrical examination of sensory functions is of but little practical importance, except perhaps when mapping out areas of loss of taste, where a mild galvanic current is an excellent gustatory stimulus.

For diagnostic purposes the three most important forms of electricity are the *faradic*, interrupted, or induced current, the *galvanic* or continuous current, and the *vibrant* electricity, which is the result of discharging a powerful induced current through a Crookes' vacuum-tube, producing the well-known X-rays. The application of this latter—so-called skiagraphy, though of great practical importance, does not specially concern the neurologist. We have to consider more particularly the faradic and the galvanic currents.

To produce the **galvanic current**, we employ a galvanic battery (preferably a dry-cell battery, which can be carried about without spilling) and this battery must have sufficient electro-motive force or voltage to overcome the resistance of the skin, which is a bad conductor, and to stimulate the muscles and nerves underneath. In cities where there is an electric light system run by the continuous current, we can utilise this current to charge an accumulator which can be carried about, or we may use the current direct from the main, provided we are careful to reduce the voltage sufficiently. This is accomplished by means of shunts, resistances, or rheostats.

Faradic, or induced electricity, is obtained by induction. In a faradic machine there are two coils of wire, concentrically placed —the primary coil within the secondary. When a galvanic current passes along the primary coil, there is produced, at the moment of closure and again at the moment of opening of the primary current, an instantaneous faradic shock in the secondary coil, no shock occurring during the period of flow of the primary current, so long as its strength remains constant. Within the primary coil there is usually a bundle of soft iron wire, which becomes converted into a magnet when the galvanic stream flows round the primary coil. Now a magnet pushed within a coil produces an instantaneous faradic or induced shock in that coil; so that as the soft iron is alternately magnetised and then de-magnetised by the primary coil, we have the effect of the magnet superadded to that of the primary coil. We can vary the strength of the faradic shocks in the secondary coil in several ways, by pulling the iron bundle in or out, or by sliding a cylindrical brass shield between the primary and secondary coils (the effect on the secondary coil being greatest when no shield is interposed), or lastly, by having the secondary coil on a sliding sledge, so that we can pull the two coils apart. This last is the best and most delicate way of varying the intensity of the faradic shocks in the secondary coil.

We apply the electrical current to muscles and nerves by means of metal electrodes of various sizes, some flat and disc-like, others with rounded bulbous ends. The metal surface of the electrode should be covered with chamois-leather. The electrodes are screwed on to a holder, which must have a contact-key whereby we can interrupt the current at will. The chamois-leather should be soaked before use, and the skin should also be well moistened, in warm water to which a little salt has been added, to render it a better conductor. The salt, however, spoils the leather, which must in consequence be frequently renewed.

We should have a galvanometer in the circuit of the galvanic

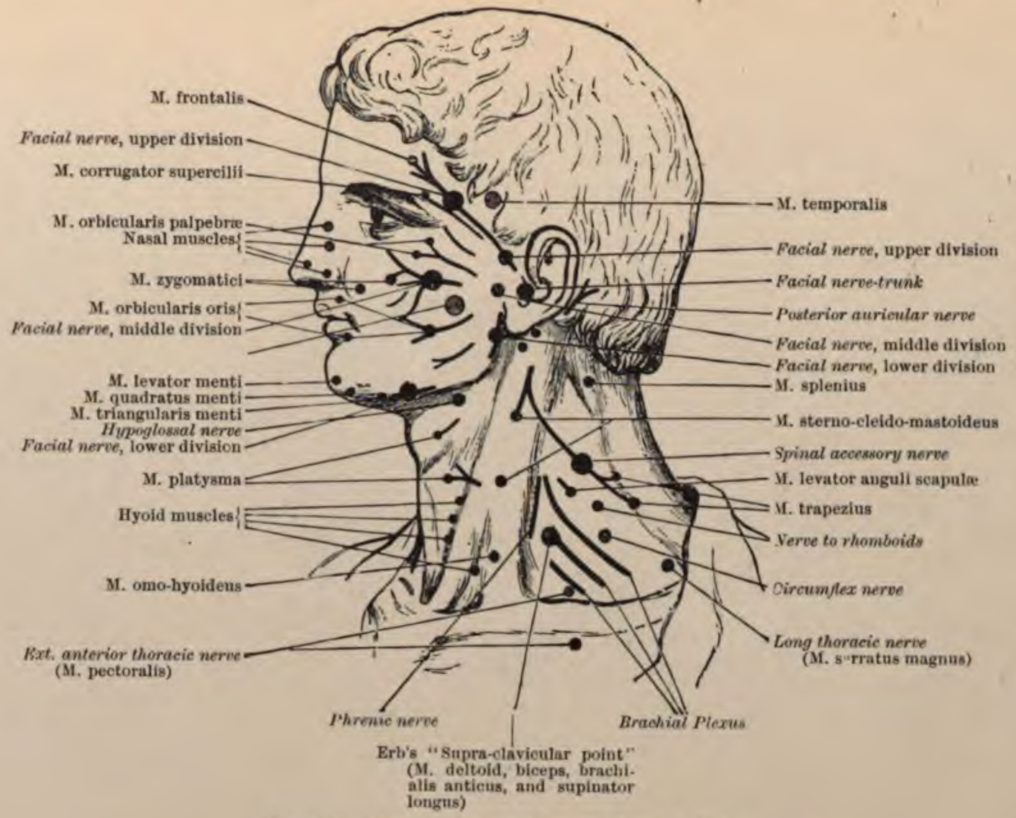


FIG. 183.—Motor Points of Face and Neck. (Erb.)

current, so as to measure the strength of current which penetrates the tissues. And there should also be a commutator or sliding switch, whereby we can reverse the direction of the current. The galvanic battery should be fitted with a collector whereby we can switch in the different cells, one by one, gradually increasing the strength of the current.

In testing the electrical reactions of muscles, one electrode should be placed on the spot we desire to stimulate, whilst the other is placed on some far-distant "indifferent" spot, where any muscular contractions that may occur will not interfere with the part we are observing. Thus the indifferent pole may be placed on the back of the neck, or on the front of the abdomen, or the patient may sit upon it, or he may hold it in the opposite hand. We then fix our attention on the other pole which we are watching. To stimulate isolated nerves or muscles, the electrode should either be a small one, or we may employ the edge of a disc electrode.

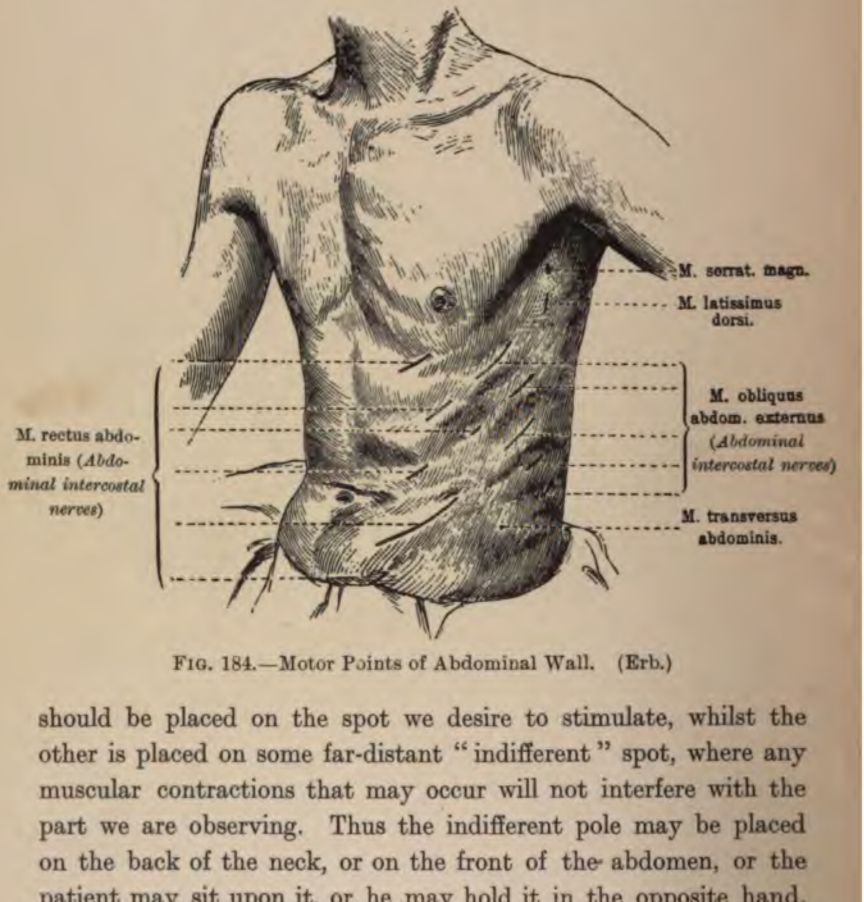

M. serrat. magn.
M. latissimus dorsi.
M. rectus abdominis (*Abdominal intercostal nerves*)
M. obliquus abdom. externus (*Abdominal intercostal nerves*)
M. transversus abdominis.

FIG. 184.—Motor Points of Abdominal Wall. (Erb.)

The patient must be placed in a good light, so that we can see the slightest movement of the muscle we are examining. Some-

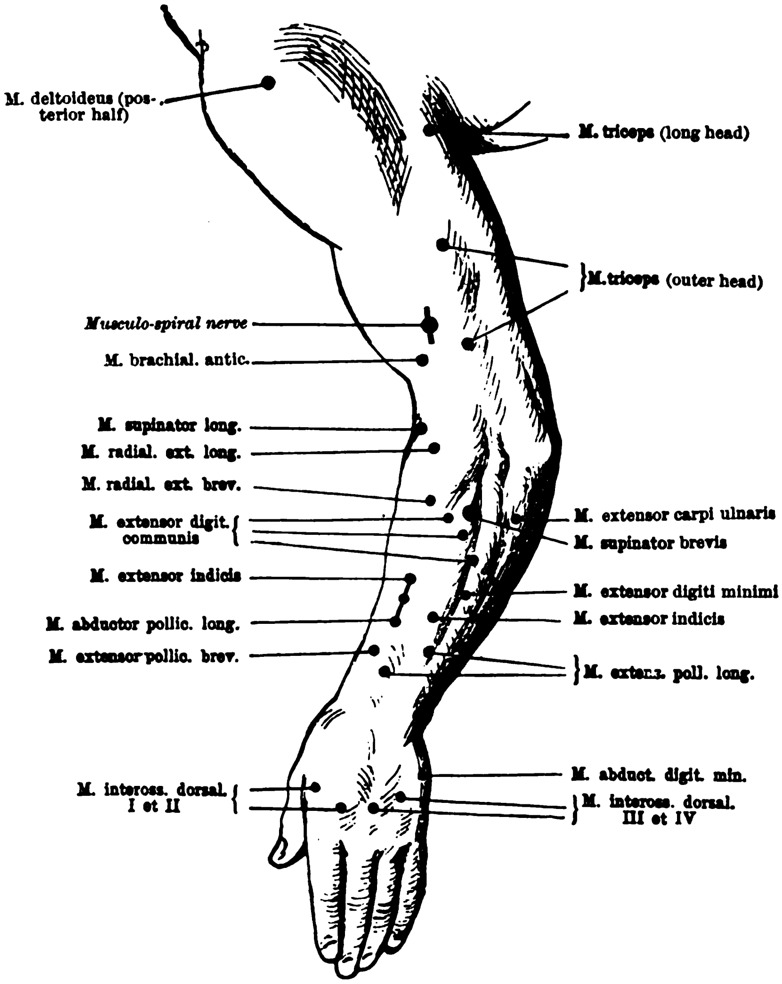


FIG. 185.—Motor Points of Upper Limb. (Erb.)

times by placing our finger on the tendon of the muscle, we can feel a contraction too faint to be visible. The patient should be made to relax all the muscles of the region which is under examination. And before applying the electrodes to the patient,

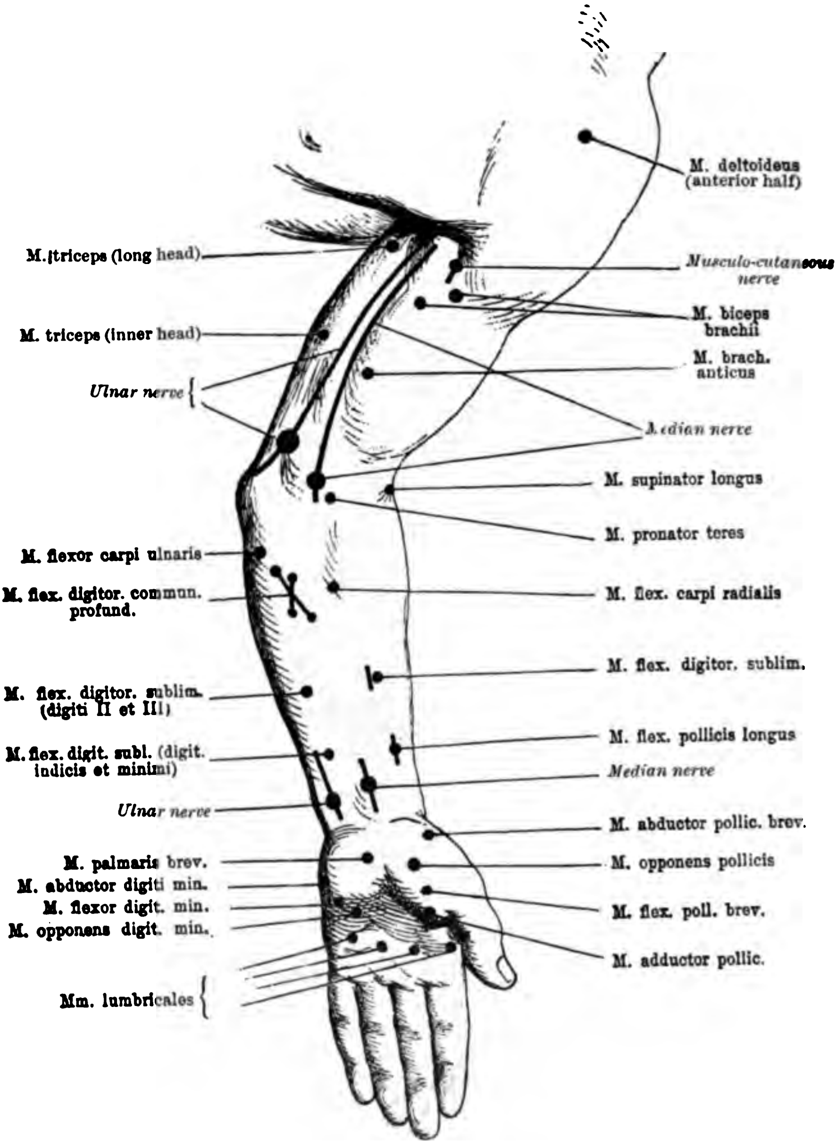


FIG. 186.—Motor Points of Upper Limb. (Erb.)

we should make it an invariable rule to test the strength of the current on our own skin, to avoid startling him by too violent a shock.

Electro-Diagnosis.—We should commence with faradic shocks. Ordinarily these are produced in rapid succession by a vibrating Neef's hammer, "making" and "breaking" the circuit of the primary coil. If these are too painful to be borne, we may often overcome the difficulty by loosening the spring of the Neef's hammer and making and breaking the primary current by moving the spring with our finger. This produces an isolated faradic shock, each time we press the spring into contact with or remove it from the screw. Such single shocks are often tolerated by a patient who cannot bear the ordinary series of shocks in rapid succession. In children, however, it is sometimes impossible to get the patient

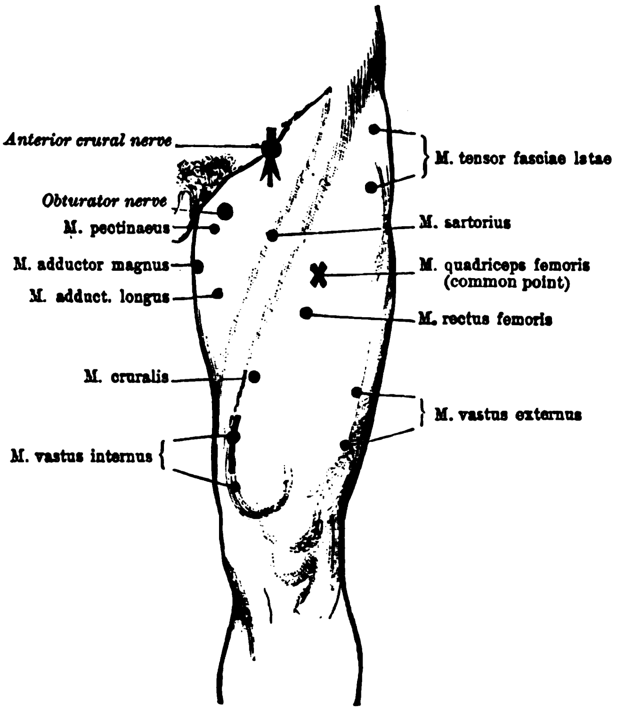


FIG. 187.—Motor Points of Anterior Thigh Muscles. (Erb.)

to submit even to single shocks, and it may be necessary in them to give a general anæsthetic, in order to make an accurate electrodiagnosis.

Whenever possible, we should compare the reaction of the suspected muscles with that of other muscles which are healthy, preferably the corresponding muscles of the opposite limb. Of course, when the disease is bilaterally symmetrical, this is impossible. An electrical examination, to be complete, would have to include observations on every accessible muscle and motor nerve in the body; this, however, is practically unattainable, and we usually content ourselves with selecting a group of muscles in the affected area and testing each muscle carefully, both with faradic

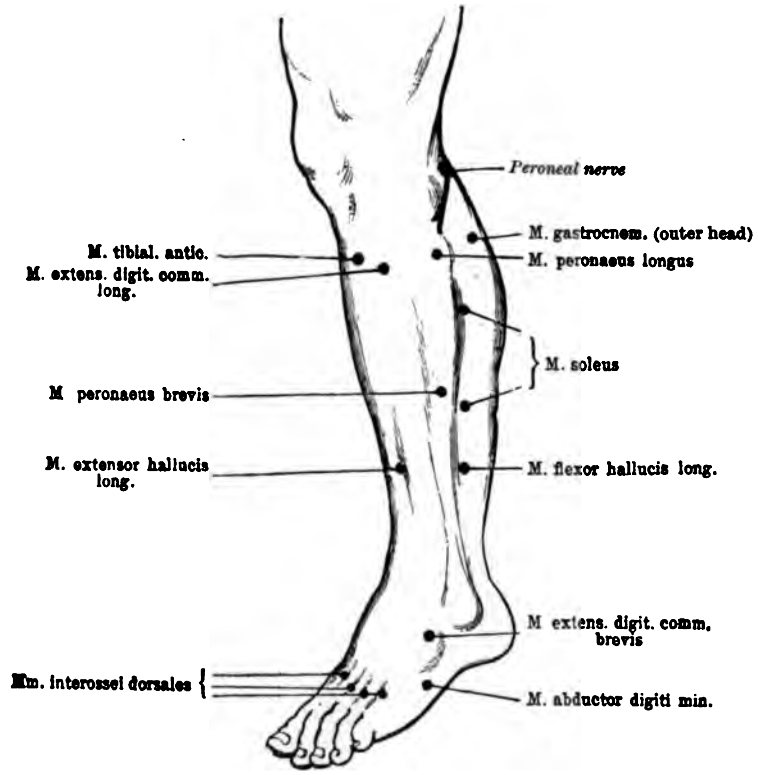


FIG. 188.—Motor Points of Leg. (Erb.)

shocks and with the continuous current, commencing with the faradic

Faradic Reactions.—Faradism stimulates a muscle most efficiently, not by direct action on the muscle-fibres but through the motor nerve. To stimulate an individual muscle by faradism we seek for the place where the nerve enters the muscle. This is usually a well-defined spot known as the "*motor point*" of the particular muscle. Faradism applied at such a "motor point" provokes a maximal contraction in that individual muscle.

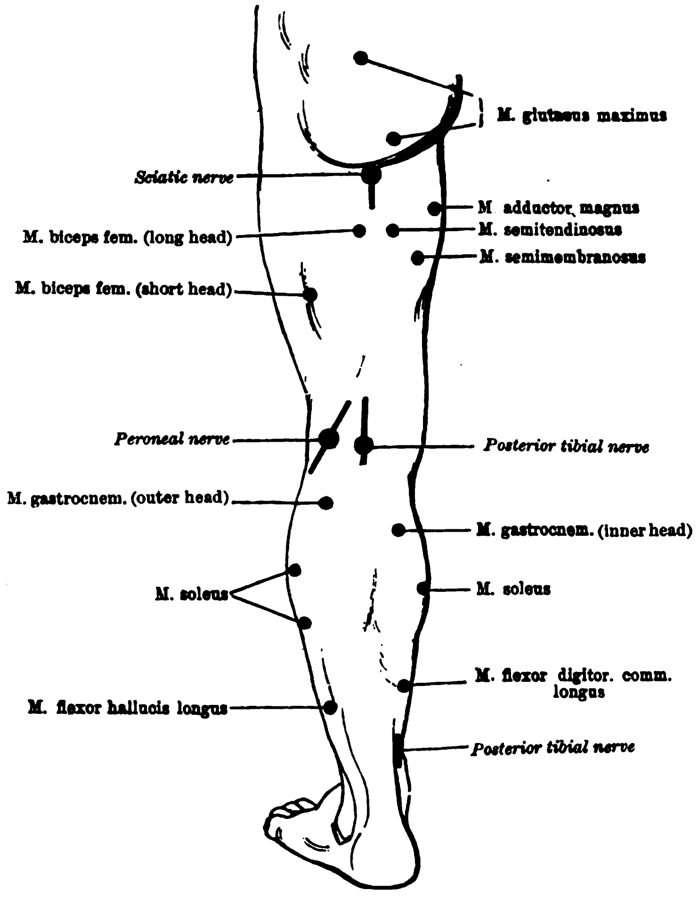

M. glutaeus maximus
Sciatic nerve
M. adductor magnus
M. biceps fem. (long head)
M. semitendinosus
M. semimembranosus
M. biceps fem. (short head)
Peroneal nerve
Posterior tibial nerve
M. gastrocnem. (outer head)
M. gastrocnem. (inner head)
M. soleus
M. soleus
M. flexor digitor. comm. longus
M. flexor hallucis longus
Posterior tibial nerve

FIG. 189.—Motor Points at back of Thigh and Leg. (Erb.)

The positions of the chief "motor points" are indicated in Erb's well-known diagrams (Figs. 183 to 189). A faradic shock of a given strength produces a much greater effect when applied at such a "motor point" than when applied directly over an indifferent bundle of muscle-fibres.

Besides stimulating individual muscles, we can stimulate whole groups by applying our electrode over a nerve-trunk, such as the ulnar or musculo-spiral.

In testing faradic reactions we commence with feeble shocks and gradually increase their strength until we just get a contraction of the muscle. We then compare this contraction with that produced by the same strength of shocks applied to a healthy muscle, preferably the same muscle of the opposite limb, if available.

Galvanic Reactions.—The galvanic current stimulates a motor nerve-fibre at the moment of closure, and again at the moment of opening of the current, but not during the period of flow, so long as the strength of the current remains constant. In the case of muscle-fibres, galvanism stimulates them at closure and again at opening, and even throughout the whole period of flow, provided the current be strong enough. Even when a nerve is degenerated, directly applied galvanism is still able to produce contractions in the muscle-fibres.

The "*polar reactions*" of a muscle to galvanism are of great clinical importance. Normally, in a healthy muscle, when we stimulate it with a galvanic current, not too strong, we get a brisk twitch at closure, then during the period of flow the muscle remains relaxed until the current is suddenly opened, when we may get another twitch at opening. The contraction at closure is greater if we stimulate with the kathode (negative pole) than if we employ the anode (positive pole). This is expressed by the formula KCC > ACC (kathodal-closure-contraction greater than anodal-closure-contraction). To verify this clinically on a healthy muscle, we gradually increase the strength of our current by means of the collector. Meanwhile, we make an occasional double movement of the commutator, whereby the testing pole is suddenly changed from kathode to anode and back again. Presently, as the current is increased in strength we find that at one position of this double movement we get a brisk twitch of the muscle, whilst in the reverse position we get none. The first twitch in a healthy muscle always appears at the kathode. Then if the current be still further increased, a twitch appears at both phases of the commutator, but the kathodal contraction remains the greater. As a matter of convenience it is best to employ a current just strong enough to give KCC, whilst as yet there is no ACC. Meanwhile we notice on the galvanometer the number of milliamperes of current which were required to produce the earliest twitch at closure.

If the strength of the galvanic current be still further increased we obtain a twitch at opening, the anodal contraction being produced first and the kathodal opening contraction last of all. The order of appearance of these different contractions in a healthy muscle, as the current progressively increases in strength, is therefore as follows: KCC > ACC > AOC > KOC, and is indicated in the following amplification of the same facts:—

1. Weak current	KCC	...	...	...
2. Medium current	KCC	ACC	...	...
3. Moderately strong current	KCC	ACC	AOC	...
4. Very strong current	KCC	ACC	AOC	KOC

Of these phenomena, we usually concern ourselves, for practical purposes, only with the first two, that is, with the contractions on closure, observing whether the kathodal closing contraction is greater than the anodal closing contraction, as it ought to be in health.

To recapitulate, in a normal nerve-muscle organ we obtain a good contraction on faradic stimulation, whilst to galvanism there is a brisk twitch on closure, KCC being greater than ACC.

Abnormalities in Electrical Reactions.—Sometimes the excitability of the nerve-muscle organ is increased, both to faradism and to galvanism. This condition of hyperexcitability is met with most typically in *tetany*, where both nerve and muscle are unduly easily thrown into contraction. Somewhat similar is the so-called "*neurotonic*" *reaction*, described by Marina in certain cases of hysteria, and by Remak in patients with progressive muscular atrophy. This consists not only in excessive excitability both to faradism and galvanism, but also in a tendency for the muscle to remain in a state of tetanus for some time after the stimulus has ceased. The phenomenon is not provoked by stimulation of the muscle itself, but only by excitation of the nerve.

We sometimes meet with simple diminution of excitability, both to faradism and to galvanism, but without alteration of polar reactions—that is to say, KCC remains greater than ACC. Such *diminution of electrical excitability* is met with in simple arthritic muscular atrophy, in the atrophy of disuse and also in the various myopathies, whether pseudo-hypertrophic or atrophic in type.

Temporary loss of faradic excitability occurs in myasthenia gravis. The *myasthenic reaction* consists in the fact that after a certain number of faradic shocks, the muscle gradually reacts less and less, until at last it shows no contraction to the strongest faradic shocks. We wait a few minutes and then test again, when we find that the faradic excitability has reappeared, but can again be exhausted in a similar fashion. The galvanic reactions of the affected muscles remain unchanged throughout the disease.

During the paroxysms of the rare disease known as *family periodic paralysis*, the paralysed muscles are, for the time, totally inexcitable either by faradism or by galvanism. In the intervals between the attacks of paralysis, the muscles react normally.

The *myotonic reaction* is met with only in Thomsen's disease (myotonia congenita). It consists in the fact that on faradic stimulation the muscular contraction persists for some time after the stimulus has ceased, as if the muscle, once contracted, could not relax. Moreover, in this disease galvanic stimulation of the muscle produces curious wave-like contractions, and KCC is equal to instead of greater than ACC.

Reactions of Degeneration.—By far the most important modification of electrical reactions is the condition known as the

" reactions of degeneration," or colloquially as *R.D.* This condition is present when the nerve-muscle organ has undergone degeneration, from disease or destruction of the spino-muscular motor neurone. As a result of such a lesion, the motor nerve-fibre disintegrates within a few days and loses its power of conducting impulses. And the corresponding muscle-fibre undergoes important changes; it loses its fibrillar or anisotropic element, the element which contracts with a brisk twitch, and which can be stimulated with a faradic shock; whilst it retains only its sarcoplasm, a less excitable element, which contracts slowly and can still be stimulated by galvanism.

In a typical case the phenomena are as follows:—to *faradism* there is no response, since the nerve has degenerated; to *galvanism* the muscle-fibres still respond—in fact after a time they become hyperexcitable, contracting to a weaker current than in health. But their polar reactions are altered. The anodal contraction on closure is now equal to, or greater than the kathodal (ACC > KCC). Moreover, what is equally characteristic, the response of the muscle is no longer a brisk twitch; it is a slow, sluggish, almost vermiform movement. If a nerve be divided, the reactions of degeneration do not appear at once. It is only after some ten days or so that they develope. Once established, the reactions of degeneration persist, unless the nerve regenerates and re-establishes a connection between the muscle and the motor nucleus. In the process of recovery, voluntary motor power reappears before faradic excitability returns. In many cases recovery does not take place and the nerve-muscle organ remains permanently degenerated, as, for example, where the motor nucleus in the cord or medulla is destroyed, or where a nerve-trunk is completely divided and its ends have not been united.

In certain cases we meet with a *partial or incomplete reaction of degeneration*. This consists in a sluggish contraction to galvanism, ACC being greater than KCC, but the reaction to faradism is not lost, but only diminished. This indicates a less severe injury of the nerve-fibres than if typical R.D. be present.

Sometimes we have *mixed reactions*, where some fibres of a muscle retain their normal reactions whilst adjacent fibres have reactions of degeneration. This is best exemplified by cases of progressive muscular atrophy where degenerated muscle-fibres are interspersed amongst the healthy.

To sum up, then, the presence of R.D. always indicates a lesion somewhere in the lower or spino-muscular motor neurone. We should be careful to wait ten days or a fortnight from the onset of the paralysis, before giving this verdict, inasmuch as we have seen that it takes some time for degeneration to become established. R.D. occurs in lesions of peripheral motor nerves, also in gross nuclear diseases such as acute anterior poliomyelitis, hæmorrhage, or thrombosis in the anterior cornua or motor nuclei. Mixed reactions, on the other hand, are found in progressive muscular atrophy and in bulbar palsy, where the nerve-cells of the motor nuclei are picked out one by one, leaving adjacent nerve-cells unaffected.

Electro-Prognosis.—In many paralyses due to organic lesions of peripheral motor nerves, of which the commonest instance is that of a neuritis of the facial nerve, it is of importance to be able to estimate not only the degree of degeneration which has occurred, but also the prospects of recovery. To make an accurate prognosis we must wait at least ten days, and preferably a fortnight, before making our investigation, so as to allow time for degenerative changes to have occurred. Electrical examination at any earlier date is practically valueless for purposes of prognosis. If, after a fortnight's motor palsy—say, in a case of Bell's paralysis—we get typical R.D., the degeneration of the nerve is severe and recovery will not commence for three months at least, possibly not for a year, and the patient may even remain permanently paralysed. And at the best, if recovery does occur, it will probably be imperfect and associated with some contracture. If a partial R.D. be present, the prognosis is less grave and recovery may be expected within six or eight weeks. If the reactions be normal, or if there is simply a diminution to faradism and galvanism, but without polar changes, recovery may be looked for in from three to six weeks, or even sooner.

LECTURE XXIII

THE CEREBRO-SPINAL FLUID

In the adult, the spinal cord terminates at the level of the lower part of the first lumbar vertebra. Below that level the arachnoidal sac extends as a hollow cavity as low down as the second sacral vertebra (see Fig. 190). There is thus a considerable extent of arachnoidal cavity devoid of spinal cord, occupied simply by the roots of the cauda equina and by the cerebro-spinal fluid which bathes them. From this region we can withdraw cerebro-spinal fluid without risk of injury to the spinal cord. In order to do so, we enter the cerebro-spinal pond from behind, somewhere between the laminæ of the second lumbar and the second sacral vertebra. The cerebro-spinal fluid, secreted by the ependyma covering the choroid plexuses, receives various products of metabolism of the nerve-centres, and may participate in lesions of these centres or of their meninges.

The two widest inter-laminal spaces are the one between the third and fourth, and the other between the fourth and fifth lumbar laminæ. Of these we usually select the space between the fourth and fifth laminæ, because, as a rule, it is slightly the larger. These two inter-laminal spaces are very easily found as follows : we draw a horizontal line across the patient's back, at the level of the highest margin of the iliac crests. This line intersects the vertebral column at the tip of the fourth lumbar spine. We make our puncture immediately below this spine (Fig. 191).

We use an all-glass syringe which is readily sterilisable, on which to mount our needle. The needle itself is made of platinum and iridium, so that it can be boiled without rusting, unlike a steel needle. The needle must be long enough to reach into the arachnoidal sac, and yet short enough to stop short within the sac without penetrating to the dura on the far side. For this purpose the most convenient length of needle is about eight centimetres, or just over three inches.

As to the patient's posture during the operation of "lumbar puncture," he may be lying on a bed or couch, in the left lateral position, with the knees and shoulders approximated. But it is better, if possible, to have him sitting on a low seat, stooping well forwards, with his knees separated, his arms hanging loose, and his hands touching the ground. In this way the laminæ are separated to their utmost extent (see Fig. 191).

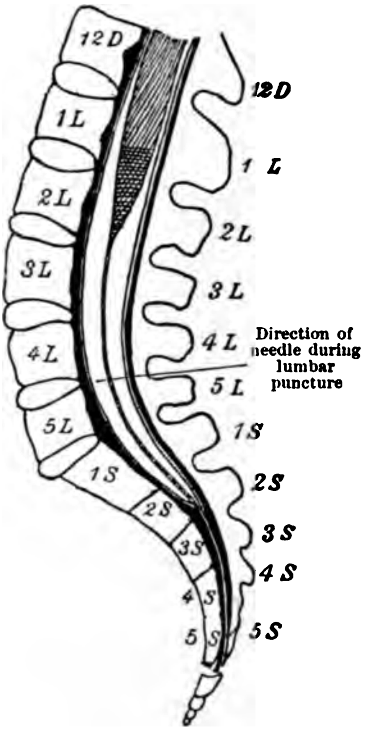


FIG. 190.—(After Raymond.) Diagram of lower end of vertebral column and its relations to the spinal cord and cauda equina. The double-shaded portion of the cord is the conus terminalis, with which the filum terminale is continuous.

We carefully sterilise the skin at the site of puncture and render it anæsthetic by means of a spray of ethyl chloride. The operator places his left index finger on the fourth lumbar spine as a guide, and with his right hand pushes in the needle, about half an inch below and half an inch to the right of this spot, so as to avoid the dense interspinous ligament, directing the point of the needle horizontally forwards and a little inwards. The ligamentum subflavum, deep in, between the laminæ, is somewhat resistant, and the needle is felt to be checked here. But we push firmly on, if no bone is struck, and suddenly the needle penetrates the ligament and dura-arachnoid and is now in the arachnoidal sac (see Fig. 192). If we strike on a lamina instead of the ligament, we withdraw slightly and try a point above or below.

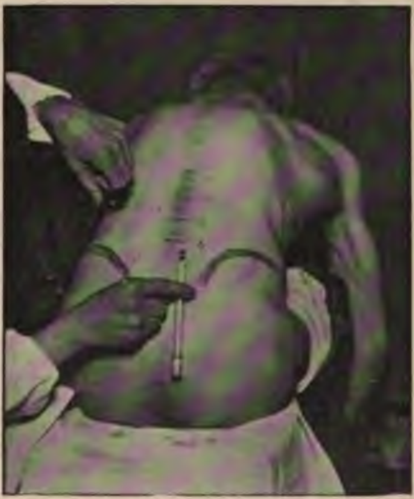


FIG. 191.—Lumbar puncture. Fluid dropping from needle into test-tube.

Sometimes during the operation the patient feels a sudden, sharp pain shooting down his right thigh and leg. This simply means that our needle has touched one of the roots of the cauda equina *en route*, and is of no other significance.

We now detach the syringe and allow the cerebro-spinal fluid to escape through the needle, collecting from 3 to 5 c.c. in a sterilised test-tube. It is better not to employ suction by the syringe, unless some difficulty be experienced in getting the fluid to run. The pressure of the fluid as it escapes may be measured, if desired, by means of a rubber tube with a manometer attached.

Normally the fluid trickles out slowly, drop by drop. The first few drops should be discarded, since they may be stained by the admixture of blood from our needle wound of the superjacent tissues.

Occasionally it happens that even when we have successfully penetrated within the ligamentum subflavum, no fluid will flow. This is usually due to the blocking of the needle by a small plug of blood-clot or shred of muscle or connective-tissue, during the process of puncture. Or it may be due to the floating of one of the cauda equina roots against the end of the needle. Such conditions are easily rectified by passing a sterilised stilette along the needle, to clear it. Sometimes it happens that the needle pierces the ligamentum subflavum but pushes the spinal theca in front of it, so that, even though the stilette shows the needle to be free from obstruction, no fluid comes. In such cases it is best to withdraw the needle and puncture at the next space above.

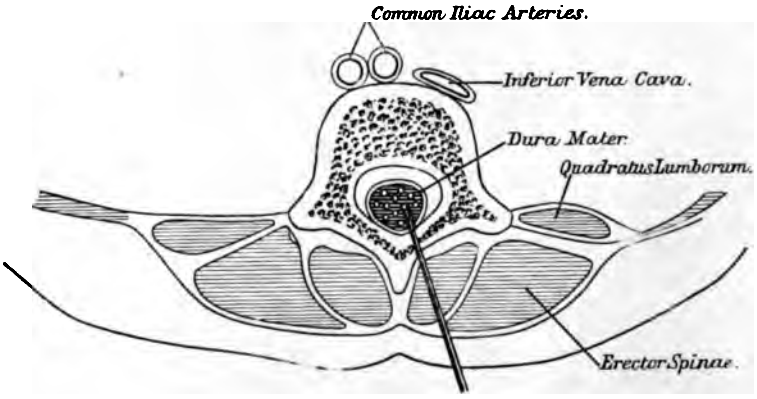


FIG. 192.—Horizontal section at the level of the fourth lumbar vertebra, showing the relations of the parts concerned in lumbar puncture.

Sometimes the intrathecal pressure is increased, so that the fluid escapes in a jet, instead of drop by drop. This occurs chiefly in intra-cranial tumours and in the various forms of meningitis, although even in these conditions an increased pressure of fluid in the lumbar region is by no means constant, for sometimes in meningitis there is occlusion of the foramen of Magendie, or of the foramina of Key and Retzius, cutting off the intra-ventricular from the spinal pond.

The fluid may be accidentally mixed with blood from the wounding of an arachnoidal vein during the puncture; this is an accident which cannot be foreseen. In most cases such bleeding clears up after a few seconds, the fluid becoming progressively clearer and clearer. But admixture of blood also occurs in cases of pre-existent subarachnoid hæmorrhage, cerebral or spinal, as in fracture of the skull, intra-ventricular hæmorrhage, or in trauma of the spinal cord. We can sometimes distinguish between hæmorrhage due to the local puncture and hæmorrhage which results from a previous intra-thecal lesion. We do this by centrifuging the fluid. In local hæmorrhage due to puncture, the blood corpuscles fall to the foot of the centrifuge-tube, leaving the superjacent fluid clear and limpid, whereas in a pre-existing cerebral or spinal hæmorrhage the fluid (which during its flow was equally tinged from start to finish, and generally less deeply tinged than in local hæmorrhage), remains of a yellow colour even after centrifuging.

The withdrawal of a small quantity of cerebro-spinal fluid in most cases causes the patient no discomfort. But in a small proportion of cases he may complain of severe headache, appearing an hour or two later. This is usually in patients who have been walking about as usual, and the headache can generally be prevented by recommending the patient to rest for a day or so, or at least to avoid physical exertion.

Normal cerebro-spinal fluid is colourless, like water. It has a specific gravity of 1006 to 1008. Chemically it is alkaline, contains a trace of serum-globulin and of albumose, and also a substance which reduces Fehling's solution. Microscopically it contains a few large, flat, endothelial plates, and perhaps an occasional lymphocyte. It contains no organisms.

The Diagnostic Significance of Cerebro-spinal Fluid.—For diagnostic purposes, the fluid may be examined in various ways.

(1) *Physical Characters.*—Instead of being clear and limpid like water, the fluid may be turbid and cloudy, or even purulent, as in some cases of meningitis. In severe jaundice it has been

observed to be yellow and clear, whilst in cases of hæmorrhage either into the brain or cord, as we have mentioned, it may be blood-stained.

In cases of sudden hemiplegia, we are sometimes in doubt whether the lesion is hæmorrhage or thrombosis. And the diagnosis between these two conditions may be very difficult. But the treatment of the two is diametrically opposite. In the case of thrombosis we stimulate, whilst in cerebral hæmorrhage we try to lower the blood-pressure and diminish the force of the heart. If in a doubtful case we perform lumbar puncture and obtain blood-stained fluid, the diagnosis may be much simplified. I have performed lumbar puncture in a comatose patient moribund from cerebral thrombosis and obtained normal clear fluid. In traumatic cases it is of even greater value in indicating the presence of intra-cranial hæmorrhage. Cathcart of Edinburgh[1] recorded a case in which, after a head injury, examination of the cerebro-spinal fluid helped to settle the diagnosis of intra-cranial hæmorrhage. He trephined, tied the ruptured artery, and saved the patient's life.

The freezing-point of the cerebro-spinal fluid has been studied by various observers:—so-called *cryoscopic examination.* Widal, Sicard, Ravaut,[2] and others have recorded a lowering of the freezing-point in certain diseases such as meningitis, tuberculous or otherwise. But, on the whole, cryoscopy of the cerebro-spinal fluid has not been of great practical value.

(2) *Chemical Characters.*—These are of some value. Thus, for example, an excess of albumin is found in acute meningitis, and in general paralysis of the insane. If fibrin is found, this also is pathological. Guillain and Paraut[3] recorded some interesting observations on the cerebro-spinal fluid in cases of general paralysis of the insane. When normal cerebro-spinal fluid is boiled, the albumin coagulates and the fluid becomes slightly opalescent. If the globulin be precipitated by adding a saturated solution of magnesium sulphate, the fluid filtered, and the clear filtrate again boiled, it should remain clear. In general paralysis

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[1] *Scot. Med. and Surg. Journ.*, Edinburgh, 1902, p. 145.

[2] *Compt. rend. Soc. de Biol.*, Paris, August 20, 1900.

[3] *Rev. Neurol.*, Paris, April 30, 1903.

of the insane, however, the second boiling yielded a characteristic precipitate in every case out of a series of sixteen general paralytics examined by these observers.

Another chemical point, which my colleague, Dr. Hebb, investigated in a number of my cases at the Westminster Hospital, was as to the presence of cholin in the cerebro-spinal fluid. Normally cholin is not detectable in any appreciable quantity. But in organic degenerative diseases of the nervous system, cholin may be demonstrated in the blood and cerebro-spinal fluid, and can be crystallised out as a combined platinum salt, according to the method of Halliburton and Mott. Thus, for example, Hebb obtained cholin-platinum crystals from the cerebro-spinal fluid in cases of cerebral hæmorrhage, syringomyelia, disseminated sclerosis, &c. This test might occasionally be of value in the diagnosis between hysteria and gross organic disease. But it is somewhat too complicated and technical for ordinary clinical work.

(3) *Bacteriological Characters.*—These are chiefly of value in cases of meningitis, to determine the particular organism causing the disease. Thus, for example, in epidemic cerebro-spinal meningitis the meningo-coccus (*diplococcus intra-cellularis*) is found; in tuberculous meningitis, the tubercle bacillus; and in other varieties of meningitis we may find staphylococci, streptococci, pneumococci, and so on. But we should remember that failure to discover tubercle bacilli does not necessarily exclude tuberculous meningitis, though their presence would confirm such a diagnosis. In doubtful cases inoculation experiments on guinea-pigs are of value.

(4) *Microscopic Characters.*—Clinically, microscopic investigation has proved by far the most valuable mode of examination of the cerebro-spinal fluid. A good routine method is to centrifuge a given quantity of the fluid, say 5 c.c. for five minutes, so as to collect into a small deposit all the cellular elements which may be present. We then carefully decant off all the superjacent fluid, turn the empty tube upside down and scrape the bottom with a fine capillary pipette. In this way we collect the sediment, which is transferred to a slide, fixed by heat, stained by methyl

blue or by Jenner's stain, mounted in Canada balsam and examined with the microscope. In rare cases we may find parasites. Thus, for example, Castellani found the characteristic trypanosome of sleeping-sickness not only in the blood of such patients but also in the cerebro-spinal fluid. This was so in every one of his thirty-two cases and also of forty cases examined by Bruce.[1] In fact, it is easier to identify the trypanosome in the cerebro-spinal fluid than in the blood, where it is likely to be obscured by blood corpuscles. In a few cases of malignant growths of the spinal cord or of its meninges, tumour-cells have been found in the fluid. But this is inconstant, though tumour-cells, if present, would have a high positive diagnostic value.

By far the most valuable point to be determined microscopically is the presence or otherwise of leucocytes, observing not only their number but their type. This is the so-called **cyto-diagnosis.**

Normal cerebro-spinal fluid contains no polymorpho-nuclear leucocytes and only an occasional small mono-nucleated lymphocyte, with now and then a few endothelial plates. Examination of the centrifuged deposit with a magnification of 400 diameters, according to the technique already described, should show an average of not more than two or three lymphocytes to the field. Sometimes we find no cells of any sort. But in certain organic diseases of the central nervous system or its membranes, there may be a large excess of leucocytes—polymorphs or monomorphs. Briefly stated, in cases of acute microbic infection of the brain and meninges, especially the suppurative varieties, we find a polynuclear leucocytosis, where the leucocytes are mostly polymorphs, with some large monomorphs as well. In acute infective meningitis, when recovery sets in, the polynuclear leucocytes in the cerebro-spinal fluid diminish in number and become replaced by lymphocytes. These latter in turn disappear as convalescence becomes complete.

If a brain abscess be present without implication of the superjacent meninges, as sometimes occurs, the cerebro-spinal fluid shows no excess of leucocytes. Examination of the fluid is therefore a valuable means of distinguishing between meningitis and brain abscess. Both in brain abscess and in meningitis a blood-count shows a great excess of polymorphs in the blood: these may number from 10,000 to 25,000 or more, instead of from 8000 to 10,000 per cubic millimetre as in health, the leucocytosis of the blood being higher, as a rule, in meningitis than in brain abscess.

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[1] *Brit. Med. Journ.*, London, November 21, 1903.

But in subacute and chronic affections of the meninges, whether tuberculous, syphilitic, or otherwise, also in certain chronic degenerative diseases of the central nervous system, we find a lymphocytosis, *i.e.* an excess of small monomorphs, sometimes accompanied by a small proportion of large monomorphs. In functional nervous diseases the fluid is normal.

Let us study some illustrative cases. One case was an example of epidemic cerebro-spinal meningitis in a man who was comatose and apparently moribund. The cerebro-spinal fluid was under excessive pressure, of turbid appearance, and showed microscopically an average of 87·3 polymorphs to the field. Within many of the leucocytes the meningo-coccus was readily distinguished. The withdrawal of about an ounce of fluid caused marked improvement in the symptoms and the patient made a good recovery. Another case was that of an officer who had chronic otitis media on the left side. He rapidly developed mental dulness and slight aphasic symptoms, with some fever. In his case the fluid was turbid, and showed no fewer than 371 polymorphs to the field. Operation was at once undertaken and an inflamed area of brain tissue was exposed in the temporo-sphenoidal lobe. No abscess was found. The symptoms rapidly subsided and the patient made a complete recovery.

As an instance where the cerebro-spinal fluid was normal may be mentioned the case of a child, in whom, as is so often the case, a deep-seated pneumonia was ushered in by head symptoms simulating meningitis. There was marked headache, some head-retraction, and squint. Moreover, an elder child in the same family had previously died from meningitis, and the parents were therefore in considerable anxiety as to the possibility of a second case. The cerebro-spinal fluid, however, showed no excess of cells.

Meningitis was therefore unlikely, and an encouraging prognosis was given, which proved correct.

With regard to the cases of lymphocytosis, the most striking results are those observed in general paralysis of the insane and in tabes dorsalis. The lymphocytosis in these two affections (which are essentially the same disease etiologically, and which clinically are not infrequently combined in the same patient) is more marked than in any other form of organic nervous disease. Thus in a series of eleven cases of general paralysis examined by me,[1] the average number of lymphocytes to the field was 127·5 per field, the lowest count in any one case being 26·6, and the highest 481. In my series of twenty-two tabetics, the lowest count was 36, the highest 316, and the average number per field for the whole series was 131·4. This closely approximates to the average for general paralysis.

A point of importance noticed in these cases of tabes and general paralysis is that not only was lymphocytosis present in every case without exception, but it was often extremely marked when the other symptoms of the disease were very slight. Thus, for example, one tabetic patient had no ataxia and the knee-jerks were still present; but the ankle-jerks were lost and the cerebro-spinal fluid had 214 lymphocytes to the field. The same applies to general paralysis. One patient with 481 lymphocytes to the field was an early case, with practically no mental symptoms, simply a slight loss of memory and a history of transient aphasia and weakness of the right hand following an attack of unconsciousness —a "congestive attack"; he had also slight fibrillary tremors of the face.

We therefore possess in the cytological examination of the cerebro-spinal fluid a valuable and apparently a constant means of recognising tabes and general paralysis in their earliest stages. If no excess of lymphocytes is present, these two diseases can be confidently excluded.

A further point of interest is that the lymphocytosis in tabes and general paralysis is uninfluenced by the most energetic anti-specific treatment. In several of my cases, the fluid was examined at intervals of a month, the patient in the interval having had daily hypodermic administrations of mercury; yet the lymphocytosis remained unchanged in its intensity. This is in marked contrast to what occurs in active syphilitic lesions. Thus, for example, in a case of gumma of the spinal cord, the lymphocytes, which numbered 52·6 on admission, were reduced after three weeks' treatment to 16·1, and the patient, who was totally paraplegic and anæsthetic in the lower limbs on admission, was discharged in a couple of months able to walk, without anæsthesia and with the sphincters under control.

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[1] *Edin. Med. Journ.*, 1906, p. 429.

Another point of importance is that syphilis by itself, unless an active syphilitic lesion attacks the central nervous system, produces little or no excess of lymphocytes in the cerebro-spinal fluid. Thus in my series of twelve cases, in the five secondary cases the average number of lymphocytes was 2·7, that of the six tertiary cases was 0·9, the twelfth case being one of a man who had a syphilitic chancre twenty-six years before. The tertiary cases with active syphilitic lesions, such as gummatous ulcers, rupia, and so on, had more cells than the others, but even in them the number was less than the average of the secondary cases.

We thus see that the presence of a large lymphocytosis in a patient who has had syphilis, is of grave significance and indicates serious organic disease, either syphilitic disease of the central nervous system, or, if very marked, tabes or general paralysis. In such cases we should carefully examine for other signs of organic disease, especially for Argyll-Robertson pupils, early optic atrophy, alterations in the reflexes, and sensory changes. And even if no other sign of organic disease be present, the presence of lymphocytosis of the cerebro-spinal fluid is an indication for the most energetic anti-specific treatment, in the hope, perhaps, of preventing subsequent developments.

The presence of lymphocytosis in tuberculous meningitis is readily understood, also that which occurs in tuberculous tumours on the surface of the brain. It is more difficult at present to account for the occasional excess of lymphocytes in cerebral neoplasms. Thus one case of glioma of the centrum ovale had no fewer than 75 lymphocytes to the field, a count which led one to expect a tuberculous mass, there being no possibility of syphilis. And yet the autopsy showed a glioma which had in no way approached the meninges, and where a hæmorrhage into the substance of the tumour ultimately caused death by bursting into the lateral ventricle.

Therapeutic Applications of Lumbar Puncture.—Lumbar puncture was originally introduced by Quincke for the purpose of relieving intra-cranial pressure in tuberculous meningitis. This it does for the time, though it cannot, of course, be more than a palliative measure. In one case of mine, however, the patient, a young man, comatose and apparently moribund, regained consciousness for a day, after the withdrawal of 22 cc. of cerebro-spinal fluid. At the time of puncture the fluid was allowed to flow until the increased intra-thecal pressure fell to normal. The importance of this procedure, not only on sentimental but possibly on medico-legal grounds, is obvious.

In other forms of meningitis associated with a polynuclear leucocytosis (apart from septic cases secondary to bone diseases), especially in epidemic cerebro-spinal meningitis, good results have been obtained by the injection of antiseptics, after withdrawal of a corresponding quantity of cerebro-spinal fluid. For this purpose, a 1 per cent. solution of lysol has been found satisfactory, 10 cubic centimetres being introduced at each sitting.

In intra-cranial pressure due to other causes, as, for example, in cerebral tumours which are inoperable or inaccessible, lumbar puncture is sometimes of distinct benefit by diminishing the pressure and thereby relieving headache, vertigo, and other symptoms. Hitherto to relieve intra-cranial pressure and to diminish optic neuritis it has been the custom to trephine the skull as a palliative measure. But lumbar puncture is quicker, simpler, and less dangerous than a major cranial operation. I have performed it in three cases of intra-cranial tumour where the symptoms pointed to deep-seated cerebellar disease. In all of them the headache and vertigo were greatly relieved for several weeks after the puncture, and an opportunity was afforded of studying the patient's focal symptoms with a view to subsequent removal of the growth. In intra-cranial growths, however, we must be careful not to withdraw too large a quantity of cerebro-spinal fluid, lest sudden diminution of pressure cause a hæmorrhage into the tumour (a result, by the way, which sometimes occurs after a palliative trephining). In a case recorded by Masing [1] this actually occurred; but in his case the fluid was allowed to run for quarter of an hour and no less than 10 cc. were withdrawn. I have had under my observation several cases of persistent tinnitus and giddiness, of many months' duration, in which a single lumbar puncture with the removal of less than 10 cc. of fluid permanently relieved the condition. We must, of course, be careful to eliminate all the ordinary causes of tinnitus and giddiness, aural or otherwise, before resorting to this treatment. In fractures of the base of the skull, the coma may be relieved in a remarkably short time by the withdrawal of cerebro-spinal fluid. I saw such a patient after a carriage accident, in whom lumbar puncture was followed by rapid improvement. The procedure may be repeated several times on successive days, if necessary.

Cases of uræmic coma and convulsions, and of puerperal eclampsia, are often relieved in a striking manner by lumbar puncture, and in several instances the patient's life has undoubtedly been saved by this means. For example, McVail [2] records two cases of acute nephritis in which, notwithstanding energetic treatment by purgatives, hot-air baths and pilocarpin to induce free perspiration, coma and convulsions intervened. Lumbar puncture was performed, 6 or 8 drms. of cerebrospinal fluid were withdrawn, and within three or four hours the coma passed off, the convulsions ceased, and both patients ultimately recovered completely. Cases such as these raise the question as to whether the headache, coma, and convulsions in nephritis are really caused entirely by "uræmic poisoning" of the brain-centres, or whether they may not be largely due to a sudden increase of intra-cranial pressure—part of the general œdema.

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[1] *Neurol. Centralbl.*, 1904, p. 1116.

[2] *Brit. Med. Journ.*, London, 1903, vol. ii.

Again, in cases of tetanus, we know that the tetanus poison has a selective action on the motor nerve-cells of the spinal cord and brain. And therefore, in addition to removing the tetanus bacilli at the site of inoculation, by excision of the original wound, we endeavour to neutralise the toxin by means of antitoxin. This is sometimes administered hypodermically; but it is much more efficacious, as Roux and others have shown, if injected into the cranial cavity through a small trephine opening, and some remarkable recoveries have followed this method of treatment. But the procedure is not free from danger. In one case at least, the patient died of cerebral abscess at the site of the trephine opening eight weeks afterwards, long after all symptoms of tetanus had disappeared. It is easier and better to administer the antitoxin (with perhaps the addition of β eucaine and morphine) by means of a lumbar puncture needle. Strychnine poisoning is also successfully treated by intra-thecal administration of eucaine. It may be necessary to give a general anæsthetic before the first intra-spinal puncture, to relax the opisthotonos.

Within the last few years, injection of cocaine or of stovaine by lumbar puncture has been a somewhat popular procedure amongst surgeons, to induce spinal anæsthesia for the purpose of performing operations on the lower limbs or the lower part of the trunk. The patient in these cases remains conscious throughout the major operation. Such a procedure, however, should be reserved for very special cases, such as acute abdominal conditions where a general anæsthetic is particularly dangerous, since, from the patient's point of view, complete unconsciousness is preferable to the mental strain of remaining conscious whilst an operation is being performed on his anæsthetic legs. Moreover, there is a risk of the spinal anæsthetic ascending too high, to the medulla and paralysing the vital centres there. This has actually occurred in some cases.

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